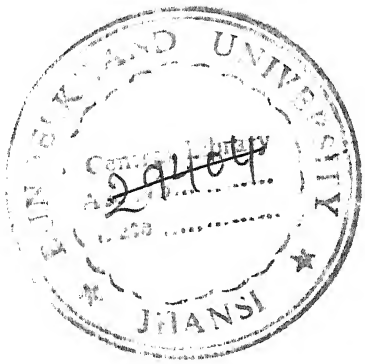
ELECTROENCEPHALOGRAPHY AND EPILEPSY IN CEREBRAL PALSY

THESIS

FOR

DOCTOR OF MEDICINE

(PAEDIATRICS)



BUNDELKHAND UNIVERSITY

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RAVI PRAKASH AGARWAL

"I that am curtailed of this fair proportion,

Cheated of features by dissembling Nature,

Deform'd unfinish'd, sent before my time

Into this breathing world, scarce half madeup,

And that so lamely and unfashionable

That dogs bark at me as I halt by them."

Richard III,(Shakespeare)

C E R T I F I C A T E

This is to certify that the work entitled "ELECTROENCEPHALOGRAPHY AND EPILEPSY IN CEREBRAL PALSY" which is being submitted as THESIS for M.D. (Paediatrics) examination, 1989 of Bundelkhand University by RAVI PRAKASH AGARWAL, has been carried out in the department of Paediatrics.

He has put in necessary stay in the department according to University regulations.

Dated: 10.9.88.

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C E R T I F I C A T E

This is to certify that the work on "ELECTROENCEPHALOGRAPHY AND EPILEPSY IN CEREBRAL PALSY" which is being submitted for M.D. (Paediatrics) THESIS by RAVI PRAKASH AGARWAL has been carried out under my supervision and guidance in the department of Paediatrics. The techniques embodied in the thesis were undertaken by the candidate himself and the observation recorded have been regularly checked by me .

He has fulfilled necessary requirements of the stay in the department for the submittion.

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C E R T I F I C A T E

Certified that the work entitled "ELECTROENCEPHALOGRAPHY AND EPILEPSY IN CEREBRAL PALSY", conducted by RAVI PRAKASH AGARWAL, was carried out under my guidance, by the candidate himself.

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Certified that the work entitled "ELECTRO-ENCEPHALOGRAPHY AND EPILEPSY IN CEREBRAL PALSY", conducted by RAVI PRAKASH AGARWAL, was carried out under my supervision and guidance, by the candidate himself.

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C O N T E N T S

INTRODUCTION

INTRODUCTION

Cerebral Palsy is defined as a non progressive, neuromuscular disorder of cerebral origin and includes a group of heterogeneous disorders of variable severity, ranging from minor incapacity to total handicap. In India, it is the second commonest cause of crippling in childhood, after polio; with an approximate incidence of 1-2 per 1000 population. This means that nearly 1 baby out of every 200 live births is born with brain damage. In a survey it was observed, that out of every 1000 patients attending out patient department of pediatrics, nearly two were suffering from cerebral palsy (Prabhakhar and Kumar 1983).

The incidence of cerebral palsy is increasing at an alarming pace, in developed, as well as developing or underdeveloped countries (Brown and Fulford 1984). Two basic reasons are responsible for this rising incidence, in developing or under developed countries - firstly the poor obstetric facilities available in rural and remote areas, where even the mild obstetric complications could not be managed suitably; and

secondly the improving standard of neonatal nurseries in affluent cities where more and more low birth weight and sick babies are being saved. But in developed countries, latter reason is solely responsible for rising incidence (Brown and Fulford, 1984).

First published study relating to cerebral palsy, dates back to late eighteenth century, when Little, in his historical article held the obstetric factors responsible, for the causation of cerebral palsy (Little, W.J., 1961). Since then several obstetric and perinatal factors had been discussed as the causative or predisposing factors; the important ones are - prematurity, Birth asphyxia, Birth trauma, Hyperbilirubinemia, Hypoglycemia and LBW - babies.

This had long been a disputed point that what is the maximum age of permanent brain damage for the causation of cerebral palsy. Perlstein et al fixed the ceiling of eight years for the permanent brain damage to occur (Perlstein et al, 1955). But still later the American Academy of cerebral palsy lowered this limit to five years of age (Davis and Hill 1980).

The diagnosis of cerebral palsy is clinical. Patients present with delayed mile stones and perinatal history usually reveal some abnormality either before, during or after delivery. The neurological examination reveals upper motor neurone signs, with or without abnormal movements. Most of the patients also have some associated defects like epilepsy, mental retardation or problems related to speech, hearing, vision or behaviour.

Epilepsy is one of the commonest associated problems in patients of cerebral palsy. The incidence of epilepsy in cerebral palsy is much higher than that in general population. While 7.3 per 1000 children, in general population, suffer from epilepsy (Kaushik, A, and Sehgal, H, 1980), about 20-50% patients of cerebral palsy have epilepsy (Brown and Fulford, 1984). The diagnosis of epilepsy and its management is of paramount importance in patients of cerebral palsy as it adds further to a crippled baby. Moreover, if epilepsy is controlled further brain damage is atleast avoided (Lennox, 1942 and Waterlain, 1978). Some patients with epilepsy are misdiagnosed as suffering from other conditions and therefore not treated properly,

while some patients are diagnosed as having epilepsy when in fact they have other problems viz. pseudo-seizures and other disorders simulating epilepsy (Walton, J. 1985) e.g. Breath holding spells, stokes-adams syndrome, carotid sinus syndrome and other cardiac dysrrhythmias, syncope, migraine, vertigo, narcolepsy, rage-reaction, panic - attacks and emotional out bursts. On the other hand only abnormal behaviour, sometimes can be epilepsy. As the anticonvulsants, which are not free from side effects, if started, will have to be given for a long period, the diagnosis of epilepsy should be definite. Moreover anticonvulsant, like barbiturates further make a hyperkinetic baby worse.

Though the diagnosis of epilepsy is usually clinical but during the state of dilemma, the electroen-cephalography is the only investigative tool which may clear ones mind. Even the most sophistcated investigations of modern times like CT-scan, magnetic - Resonance - imaging and positron emission tomography are usually not fruitful. It has also been seen that prognosis of patients of cerebral palsy with abnormal electro-encephalographs, is poor, though vice - versa is not true. (Gibbs and Gibbs et al, 1963).

No published study had been performed in India which used electroencephalography as the investigating procedure, to evaluate the abnormal EEG - findings in the patients of cerebral palsy. Thinking of its common occurance, this study was performed on electro - clinical characteristics of cerebral palsy with following aims and objectives:-

1. To find out the incidence of epilepsy in cerebral palsy.

2. To find out various electroencephalographic abnormalities in patients of cerebral palsy.

3. To categorize these abnormalities as per well defined neurological subtypes of cerebral palsy.

4. To correlate the electroencephalographic abnormaltims with clinical epilepsy.

5. To correlate the Developmental - Quotient with EEG abnormalities in cerebral palsy.

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORICAL ASPECT

The history of cerebral palsy is more than a century old. In his historical treatise, William John Little (1962) for the first time gave a clear description of cerebral palsy and noted its relation with premature birth, difficult labour, and mechanical injuries, during parturition. Some earlier workers had also described cases of cerebral palsy- Andrey (1741), Delpech (1828) and Heine (1860), quoted by Collier (1924). McNutt (1895) reported 10 cases of infantile hemiplegia, who mainly showed blood on the convexity of brain in each case. Gowers (1888) thought that most cases of cerebral palsy were due to brain damage at birth. Freud (1897) considered the etiology of generalised spasticity to be difficult and premature labour. Collier (1924) thought that the "essential anatomical cause of diplegia is a primary degeneration of cerebral neurones from causes which are at present elusive."

The ancient terminology of "Little's disease, thus originated at that time. But the whole concept of this condition has undergone complete metamorphosis since then, and now the term cerebral palsy has been universally accepted to denote brain damage during the phase of its development. It was in 1930 when Wintrop M. Phelps, grouped various forms of this condition, under a common heading. He coined the term cerebral palsy to indicate that common denominator is a paralysis (Palsy) due to impairement of central nervous system (Cerebral). The old terminology of "Little's-disease" is just one aspect of related disorder and now is synonym of cerebral diplegia only (Illingworth and Polani 1958).

DEFINITION

Several attempts had been made to define the complete syndrome of cerebral palsy but none had been successful, due to diverse etiologies and menifestations. This had long been a disputed point that what is the maximum age of permanent brain damage which can lead to cerebral palsy or in other words, till what age does the brain grow as cerebral

palsy occurs during the phase of brain's development only. Perlstein et al. (1956) set the limit of eight years, earlier which if any thing goes wrong with maturing brain may terminate into cerebral palsy. However the American academy of cerebral palsy has arbitarily fixed years as the upper limit for brain damage to occur (Down and Hill, 1980).

Low and Carter (1982) defined cerebral palsy as a group of diverse non- progressive syndromes affecting brain, menifesting as impairment in motor functions and presumed to have had their onset during gestation, during parturition or in childhood. According to Prabhakar and Kumar (1983) cerebral palsy is a persistent but not unchanging disorder of posture and movement due to dysfunction of brain, present before its growth and development are completed. However cerebral palsy is not a static condition and its clinical pattern changes as brain maturation continues throughout the childhood, resulting in dynamic clinical pattern despite a static pathology (Brown and Fulford, 1984).

INCIDENCE AND PREVALENCE

Cases of cerebral palsy constitute 0.68% of the total admission to pediatric ward and form 0.2% of total out patient pediatric cases in India (Srinivasan et al., 1975).

The incidence of cerebral palsy is highly variable as it depends entirely upon the availability of obstetric facilities, socio-economic and cultural status of the population under study. Its incidence is increasing now-a-day, basically due to two reasons- Firstly improving standard of neonatal intensive care units all over the world and Secondly paucity of proper obstetric care in remote areas of under developed and developing countries (Brown and Fulford, 1984).

Its incidence varies from 0.6 to 6.0 per 1000 live births according to various studies. According to a report of Ciba Foundation (1978) the prevalence rate of cerebral palsy in Britain is 1-2.4 per 1000. The overall average incidence is around 2.5 per 1000 live births (Brown and Fulford, 1984). Calculating at

an average rate of 2 per 1000 live births, with 21 million births every year in our country, nearly 42,000 new cases of cerebral palsy are added every year to our population (Prabhakar et al., 1983).

The condition is said to be slightly more common in males and male: female ratio is 58:42, but spastic paraplegia is more common in females, with male:female ratio of 47:53. This difference could be because of the fact that spastic paraplegia is very common in prematures who are more commonly females (Perlstein et al., 1955). In India the sex ratio reported by Sharma et al., (1981) was 50:23.

CLASSIFICATION

Since the time of advent, attempts are being made to classify various types of presentations of cerebral palsy. First attempt in this direction was made by perlstein et al., whose classification with relative frequency is as follows (Perlstein et al., 1955).

A. Spastic- Type	----	68%
Hemiplegia	----	55%
Quadriplegia	----	24%
Paraplegia	----	17%
Triplegia	----	3.7%
Monoplegia	----	0.3%
B. Dyskinetic Type	----	28%
C. Atactic Type	----	4%

Another detailed classification was produced just one year later by Balf and Ingram (1956):-

Neurological - Diagnosis	Extent
1.Hemiplegia	-Right -Left
2.Bilateral Hemiplegia	-
3.Diplegia	
-Hypotonic	-Paraplegic
-Dystonic	-Triplegic
-Spastic/Rigid	-Tetraplegic
4.Ataxic Diplegia	-Paraplegic
-Hypotonic	-Triplegic
-Spastic	-Tetraplegic
5.Ataxia	-Predominantll Unilateral -Bilateral

6.Dyskinesia

-Dystonic
-Choreoid
-Athetoid
-Tension
-Tremor

-Monoplegic
-Hemiplegic
-Triplegic
-Tetraplegic

7.Mixed

The most universally accepted classification is that produced by American Academy of cerebral palsy (Down and Hill, 1980):-

A-Type of Motor disorder -

(i) Spasticity
(ii)Rigidity
(iii)Athetosis
(iv)Ataxia
(v) Tremor
(vi)Hypotonia
(vii)Mixed

B-Anatomical Distribution -

(i) Tetra plegia
(ii)Paraplegia
(iii)Triplegia
(iv)Double Hemiplegia
(v)Hemiplegia
(vi)Monoplegia

C-Degree of Severity -

(i) Mild

(ii)Moderate

(iii)Severe

The distribution of different types of cerebral palsy had been quite variable. Soon after Perlstein et al., 1955 (above), Salomonsen and Skat-vedt produced following figures (Salomonsen and Skatvedt., 1955) -

1- SPASTIC		55.3%
	Hemiplegia Monoplegia	20.3%
	Others	35%
2- Pure ATHETOSIS		14.1%
3- Pure Ataxia		5%
4- Mixed		25.6%

The distribution pattern reported by Srinivasan et al., reveals that Diplegia is the commonest pattern

1- Diplegia	62.7%
2- Atonic Diplegia	7.5%
3- Hemiplegia	20%
4- Dyskinetic	3%
5- Minimum Brain dysfunction	6.8%

Later Sharma, M. et al. reported same pattern of distribution but with slight different figures (Sharma, M. et al., 1981):-

1- Spastic	82.7%
-Diplegia	45.3%
-Hemiplegia	34.8%
-Others	19.9%
2- Athetoid	4.1%
3- Ataxic	0.9%
4- Atonic	5.0%
5- Mixed	7.3%

ETIOLOGY

As it is well known that incidence of cerebral palsy is correlated with abnormal-pregnancy/delivery/birth, the etiology may be classified into prenatal, natal and postnatal causes which are as follows (Garg and Srivastava, 1965).

A- PRENATAL FACTORS - These are responsible for about 30% cases.

(i) Genetic factors are-Hemolytic disease of new born and Kernicterus, hereditary paraplegia, Familial Tremors, Hereditary athetosis, Familial spastic

paraplegia, Familial paroxysmal choreoathetosis.

(ii) MATERNAL AGE: Mitechell (1959) found cerebral diplegia to be commoner in children of younger mothers and athetosis and mixed type being commoner in children of older mothers.

(iii) PARITY: Skatvedt (1958) found a very high incidence of cerebral palsy in first born and similar was the observation of Garg and Srivastava (1965). But Eastman and Deleon (1955) negated such a relationship.

(iv) PREMATURITY: Prematurity stands out to be the single most common factor in the causation of cerebral palsy. Eastman et al. (1955), Churchill (1974) and Prabhakar et al. (1983) reported that the incidence of prematurity in cerebral palsy is three to five times, more common than the general population. They all observed that nearly 35% cerebral palsied children are preterm at birth. A premature baby is more liable to birth injury, partial separation of placenta, and other types of antepartum hemorrhage may lead both to premature labour and foetal anoxia thus terminating into cerebral palsy (Garg and Srivastava, 1965).

(v) MATERNAL-ILLNESS: Garg et al. (1965) reported various antenatal problems like pre-eclampsia (16.9%), antepartum hemorrhage (11.3%), hyperemesis, vaginal discharge etc. (27.2%) Faber (1947) also reported about 15% cases with history of antepartum hemorrhage.

(vi) MULTIPLE BIRTHS: An increased incidence of multiple pregnancies has been reported by Asher et al. (1950) 5.4%, Shyh Jong (1953) 9%, Greenspan et al. (1953) 7.0%, Skatvedt (1958) 6.7% and Garg et al. (1965) 5.6%.

B- NATAL CAUSES - Responsible for about 60% (majority) cases. Abnormality of labour is quiet common in cerebral palsied patients, like prolonged labour, Breech delivery, Cord Prolapse, transverse, lie, Caesarian section, precipitate labour. Association of cerebral palsy with these factors had been reported by various workers - Asher et al. (1950), Denhoff et al. (1951), Lilien field et al. (1955). 44% cases in Skatvedt (1958) series had history of abnormal labour while Garg et al. (1965) record such abnormality in one third of their cases.

In the neonatal period, common abnormalities are-asphyxia, cerebral irritation, Jaundice, convulsions and feeding problems, (Garg et al., 1965). Schreiber (1940) noted 70% cases due to Birth Asphyxia. Anderson (1952) stated that a third to a half of all cases of cerebral palsy had evidences of anoxia at Birth. Eastman et al. (1955) found foetal-distress to be four times commoner in cerebral palsy than in controls. Garg et al. (1965) reported history of abnormality in neonatal period in 56.4% of their patients.

C- POSTNATAL-CAUSES: - Important postnatal problems associated with cerebral palsy are meningitis, encephalitis, Kernicterus. The incidence of cases of postnatal origin is somewhere between 10% (Perlstein et al., 1952) and 20% (Illingworth, 1958). Mitchell (1959) recorded 12% such cases and Garg et al. (1965) observed 25% patients due to postnatial causes.

Neonatal jaundice, kernicterus and athetosis have got a important correlation (Garg et al.,1965) Rh. incompalibility was observed in 17.5% cases by Brandt et al. (1958) and in 5.9% by Martin (1960). But cases of Rh in-compatibility are very few in India due to very low incidence of Rh (D)- negative

individuals in our country (Garg et al. 1965). Circulating unconjugated Bilirubin in blood in the patients of hyper bilirubinemia gains access to blood brain barrier and deposites in Basal Ganglia and cerebellum. Anoxia further accentuates bilirubin toxicity (Diamond-et-al., 1966).

PATHOLOGY

With the exception of spastic hemiplegia, the lesion in most of cases of cerebral palsy is probably deeply situated in the centre of brain, in the form of dilated III- ventricles as seen in pneumo-encephalograms (Skatvedt, et al., 1955). The intra cerebral hemorrhage (Subependymal and intraventricular) is most important factor in spastic diplegia of prematurity (Churchill et al., 1974, Brann, 1986). Three type of basic defects have been described which either act alone or in combination (Skatvedt et al., 1955).

(i) Cerebral malformations of genetic origin.

(ii) Cerebral developmental inhibition caused by damage to fetus in embryonic period.

(iii) Cerebral damage incurred during process of birth i.e. anoxic brain damage.

Pathological studies on the other hand have resulted in careful discription of various cerebral abnormalities in patients with non pregressive neurologic disorders and have led to attempts, often highly speculative, at formulating their cause. A combined clinical and pathological approach has demonstrated, however, that a given neurologic deficit can result from a cerebral malformation of prenatal origin, a destructive process of perinatal or early postnatal onset or malformation and perinatal trauma acting in concert various. Pathological changes seen are (Menkes, 1980)--

(a) Wide spread transneuronal degenerations, which may be bilaterally symmetrical, involving thalamus and several brain stem nuclei viz. inferior colliculi, superior olive and lateral lemniscus.

(b) Periventricular-encephalomalacia - Bilateral necrosis in periventricular distribution which is accompanied by astrocytic and microglial proliferation, ependymal loss and multifocal subcertical degeneration.

(c) Water shed Infarction - due to sudden arterial hypotension causing cortical infarction of those areas which are supplied by most peripheral

branches of three large cerebral arteries.

(d) Ulegyria and status Marmoratus of Basal Ganglia - This type of lesion is principally seen in full term asphyxiated babies. The lesions are located mainly in peripheral and dorsal areas of cerebral cortex, involving necrosis of gyri at the depth of sulci and the neuronal nuclei of basal ganglia and brain stem (Brann, 1986).

ASSOCIATED HANDICAPS

The child with damage to the motor mechanisms would be expected to have damage to other parts of the brain as well. Thus it is accepted that the child with cerebral palsy may suffer from a wide spectrum of other neurological disorders since cerebral palsy is merely one menifestation of brain damaged child (Brown and Fulford, 1984). Thus these patients may have associated - mental retardation, epilepsy, visual-defects viz. hemianopia squint, myopia, optic atrophy, cortical-blindness, visuo-spatical and visuo-motor problems, gaze, palsy, Hearing defects, speech defects, learning problems, difficulties of cognitive functions, Behavioural problems like reversal of sleep pattern, irritability, slow at feeding, poor concentration span, decreased threshold for fight and flight; sensory problems like astereognosis, sensory in-attention and problems of

communication (Brown and Fuiford, 1984, Garg et al., 1965).

Out of all these handicaps, the most significant is epilepsy as it is amenable to treatment. Epilepsy in cerebral palsied children is due to cerebral trauma (Woods, 1957). The over all incidence of epilepsy in cerebral palsy varies from 15% (Pirrie, 1957) to 68% (Yannet, 1944). This figure does not include convulsions due to other problems, like febrile convulsions. While only 7.3 per 1000 children, in general population suffer from epilepsy (Kaushik et al., 1980). Gauger A.B. (1951) observed epilepsy in 43% of cerebral palsied children while Skatvedt, et al., (1955) 20.6%, Perlstein et al. (1955) 47% and Garg et al., (1965) reported that 23.4% patients in their series experienced epilepsy, in their life.

The correlation of type of cerebral palsy and incidence of epilepsy is disputed. Sone workers (Gibbs and Gibbs, 1955) reported highest incidence in atonic diplegia (69%) while Aird and Cohen (1950), Perlstein et al. (1955) and Gibbs et al. (1963) observed

that spastic are more commonly aflected by seizures. They reported the figures of 65%, 57% respectively. But it is well known that epilepsy is uncommon in athetoid and diplegic patients (Gibb and Gibbs 1955), Gibbs et al., 1963 and Garg et al., 1965). According to Gibbs and Gibbs (1955) seizures are more common where arms are more commonly affected.

According to type of seizure, all the workers had reported that grand mal type seizures are the commonest, though incidence reported by them vary. The distribution of epileptics in series of Perlstein et al. (1955)- was 53% grand mal type, 24% jacksonian type, 14% generalised type other than grand mal, 7% Myoclonic type 1.6% petit mal and rest 0.4% had psychomotor epilepsy. They also compared these figures with incidence of types of seizures in general population and observed that petit mal seizures were commonest (60%), followed by Grand mal (25%), generalised but other than grand mal (10%), jacksonian seizures (3%), Infantile myoclonus (1.5%) and psychomotor epilepsy (0.5%). The corresponding figures reported by Garg et al. (1965) are 69% grand mal, 20.7% petit mal and 10.3% jacksonian type of seizures, in patients of cerebral palsy.

ELECTROENCEPHALOGRAPHY

The electroencephalography is a continuous measurement of the constantly varying electrical potential difference between any two points on the scalp or between one point and an inactive reference electrode. First human EEG- recording was published by Hans Berger (1929) although animal recordings had been made long before by Caton (1875) and Beck (1890), (Driver et al., 1982). The basic physiology underlying the EEG is largely unknown, but the electrical activity is probably derived primarily from post synaptic potentials in the dendrites of cortical neurones (Kiloh, 1972). The EEG has proven to be a useful tool for investigating specific problems of theCNS. First recording of human epileptic activity was published by Gibbs, Davis and Lennox in 1935 (Driver et al., 1982).

Pediatrics is an area in which EEG has much to contribute but in which a sound knowledge of the range of EEG norms seen in normal children is an essential prerequisite to interpretation. Such profitability has led to a proliferation of machines and several types of machines viz. 8, channel or 12 or 16 or 20 channel

recorders are available in the market and some also record ECG and/or EMG simultaneously. It is customary to operate all the channels at the same senstivity and with same frequency characteristic (Kiloh et al., 1982).

The placing of electrodes for clinical electroencephalography is usually based on some anatomical Landmarks on the external surface of the skull. Its relationship to the underlying parts of brain is of great practical importance, but can not always be easily defined (Hellstrom, 1963). Several types of electrodes had been used from time to time, notably sphenoidal Needle electrodes (Rovit et al., 1960). Nasopharyngeal electrodes (Mavor et al., 1964), solder pellet electrodes embeded in bentonite paste (Taylor et al., 1969) and Nasoethmoidal electrodes (Lehtinen et al., 1970). But chloridated silver electrodes with electrode jelly or saline as the contact medium are most satisfactory for general use. Chloriding enables the electrodes to present the same resistance to current flow in either direction, so that distortion of EEG signals does not occur such a electrode is said to be reversible or non polarizable (Kiloh et al., 1982). The interface between the subject and the electrode should

introduce the minimum possible impedance and for this purpose Bentonite paste is generally used (Kiloh et al., 1982).

This problem of electrode placement has been considered by, among others, pampiglione, who suggested in 1956, a system of electrode placement and illustrated their anatomical placement in relation to external skull and the internal cavities. He quoted Mettler's Neuroanatomy to delimit the position of central sulcus and sylvian fissure. In the report of committee on methods of clinical examination in electroencephalography (1957), in which the 10-20 system of international federation was outlined, which is based on the measurement from a land mark on the skull, (Jasper, 1958). Though this 10-20 system has not received universal acceptance, but the advantages of a standardized system far out weighs its short comings.

There are some universal acceptance of the methodology of EEG recording (Kiloh et al., 1982). It should preferably be carried out in recumbent posture, the contact resistances of electrodes should be minimized. A caliberation signal, usually 100 μV, is then recorded simultaneously on all channels and the gains adjusted

until each channel gives a deflection of 1 cm. All EEG machines have an input switching unit, with a master selector, whereby the technician can choose from several prewired patterns and individual channel selectors whereby a pattern can be set up at will. These patterns of connection between electrodes and the recording channels are known as montages. In most investigations, the use of a few standard montages is sufficient, but in some cases it will be necessary for additional montage to be used. The type of montages used in EEG recording has a critical influence on appearence of a record, even when these are based on standard electrode placement. The International Federation has laid down following guidelines for design of montages (Jasper, 1958) --

(i) Recording channels should be connected in sequence to rows of equidistant electrodes that lie along antero-posterior or transverse lines.

(ii) The order of the channels, as read from top to the bottom of the recording paper, should in general be such that those recording from

right side of the head come before those recording from the left.

(iii) Channels recording anteriorly should come before those recording posteriorly.

The sense in which the recording pen reproduces the voltage fluctuations, between a given pair of electrodes depends upon the way in which the amplifier input leads are connected to them. Referring to those as lead 1 and lead 2, the convention is that when lead 1 becomes electronegative with respect to lead 2, the recording pen makes an upwards deflection. In diagrams of montages, leads 1 are drawn as full lines and leads 2 as broken lines, so that the relative polarity of a particular discharge can always be deduced. If an arrow between two electrodes is used to indicate the sense in which the channel is conneted, it should point from lead 1 to lead 2. There are basically different types of montages -- Bipolar, unipolar and Average reference (Kiloh et al., 1982).

Bipolar montages comprise of segmential linkage of channels along antero-posterior or transverse rows of equispaced electrodes (Jasper, 1958). Each channel is connected between a pair of active electrodes on the scalp and records the potential difference

between them. If a localized discharge occurs at or near the electrode common to two channels, these will deflect in opposite directions - a phenomenon known as phase reversals. This represent the presence of an underlying focus, from where the discharge is originating.

Each channel in unipolar montages records between one active electrode (lead 1) on scalp and one relatively indifferent electrode else where (lead 2). The latter is common to all channels and is known as the common reference. Various sites of this common reference are ears, nose, chin or neck etc. But unfortunately. it is virtually impossible to find a truely neutral reference, as they will pick up certain amount of electrical activity from adjacent parts of brain, and may be contaminated by a variety of non-cerebral potentials, all of which will be registered in all channels for which this electrode is reference (Kiloh et al., 1982).

In an average reference montage, each channel records between an electrode (via lead 1) on scalp and a common reference potential (via lead 2). The common reference potential is usually obtained by joining

all the electrodes on the scalp to a common point through high resistance of equal volume. This system was originally described by Offner (1950) and used by Gold man (1950), after whom it is sometimes known.

The usual standard paractice is to record at a paper speed of 30 mm/sec and to adjust master gain control so that the pen excursions fluctuate between 0.5 and 2 cms. If high voltage discharges are anticipated, the gain should be reduced to an extent that allows their wave form to be seen without distortion due to mechanical restriction of pen deflection, (Kiloh et al., 1982). A disturbed or mentally retarded child may need some sedation and for which purpose drugs used are - Quinal barbitone, phenobarbitone, Dichloral phenazone, chlorpromazine, Trimeprazine tartarate, promazine and paraldehyde (Kiloh et al., 1982). These drugs themselves also produce effect on the EEG tracing, like beta activity in the form of discrete runs or spindles and typically at a frequency of 18-24 Hz. This activity has frontocentral preponderance and more or less symmetrical (Brazier et al., 1945).

The terminology committee of International Federation (1966) suggested that the features present in EEG record should be classified into waves, activities, rhythms and complexes and that each feature should be described in terms of its frequency or period, amplitude, phase reactions, quantity, morphology, topography, reactivity and variability (Kiloh et al., 1982).

The principal objective criteria by which a record is assessed are based upon the frequency, amplitude andshape of waves, of which it is composed, upon their spatial and temporal distributions and upon their reactivity to stimulus. An EEG record consists of components of three basic kinds (Kiloh et al., 1982).

(a) Those that are fairly continuous and very often rhyth mical.

(b) Those that are transient.

(c) Those that comprise thebackground activity, upon which the two preceding kinds are super imposed.

As a matter of convenience, the EEG frequency spectrum is divided into bands that are designated as follows (Kiloh et al., 1982) —

delta	:	less than 4 Hz
theta	:	4-less than 8 Hz
alpha	:	8-13 Hz
beta	:	14 Hz onwards.

A poly rhythmic record is one in which two or more clear rhythmic components are present, whereas the term polymorphic refers to irregular activity, the individual waves of which are of variable period. A monomorphic wave of less than 80 m.sec. duration is a called a spike whereas, one that is of 80-200 m.sec. duration is called a sharp wave. The amplitude refers to peak-topeak value in μV. whenever it is desired to compare the amplitude of particular component in EEG, it is preferable that an appropriate monopolar montage should be used (Kiloh et al., 1982).

The EEG normally appears as sinusoid like wave forms of varying frequencies in which the predominating frequency may be normally modified by many factors, for example, opening and closing of the eyes, state of consciousness, and drugs. One of the abnormalities that occurs is that of wave forms that stands out from the background frequency. Focal slowing, often, but not in variably, results from a local disturbance causing

destruction of brain tissue. This abnormality is often seen in tumours, hematomas, strokes, localized infectious or contusions of brain. In contrast, focal spikes are in general, menifestations of iritative lesions or old processes taking months or years to develop. Spikes are often seen with scars or cysts or less commonly, slow- growing tumours. Focal slowing by itself or combined with spikes should, therefore make the physician think about further studies (Lewis et al., 1977).

The interpretation of EEG findings in children is often difficult because of wide range of patterns that occur normally at any one age. During neonatal period, the clinical and biochemical state of the child at the time of recording may be a crucial factor in the interpretation of results. Even so, the visual analysis of EEG data in newborns enables quite an accurate estimation of gestational age to be made in prematurity (Dreyfus - Brisae, 1970). The EEG of a wakeful, few days old baby is of relatively low voltage, seldom exceeding 50 μV and is composed of irregular and asynchronous theta and delta components.

During deep sleep, discrete bursts of generalized delta activity occur, often associated with faster components of few seconds duration, a pattern named trace-alternant by Dreyfus- Brisae (1964).

Vertex sharp waves and K- complexes begin to appear during sleep at about 6 months of age and sleep spindles may then also appear in fronto central regions, though their frequency may be slightly lower than 14 Hz, typically seen in adults. During first few months of life there is progressive and relatively rapid increase in voltage of low frequencies in alert state and a tendency for these to become more rhythmical. The dominant frequency also gradually increases with age.

At about 18 months, intermittent low frequency alpha components may begin to appear and there may be quite a marked reduction in amount of occipital activity on eye opening. During 2-6 years the EEG is usually polyrhythmic, different components waxing and waning independently of each other. Even some degree of asynchrony or asymmetry may be normal upto the age of 5 years. Theta rhythms are present mainly at central,

temporal and posterior temporal regions. There is a tendency for all components to become more evident over posterior part of head as the child matures. From 5-15 years of age the alpha frequency increases from 8 to about 10 Hz in average subject. It cannot be emphasized too strongly that this outline of EEG maturation from infancy to adolescence is but an average picture, that the rate of evolution will vary from child to child and that many children will depart from it in any age group.

EEG IN CEREBRAL PALSY

Electroencephalographic abnormalities in the patients of cerebral palsy are very common. First attempt in this direction was made by Perlstein et al., (1946) who recorded EEG of 212 consecutive cases of cerebral palsy. They reported that the incidence of EEG- abnormality was 82% in seizure group and 45% in non seizure group of cerebral palsied children.

Still later, in 1951, Aird and Cohen performed EEG recording of 187 patients and they observed that 85% of spasties and 60% of athetoid patients had essentially abnormal electroencephalograms. Out of these, the

incidence of focal abnormality was 62% and 32% in spastic and athetoids respectively. They also observed that more severe the clinical involvement, more are the chances of EEG abnormality. However the chances of EEG-abnormalities are higher in the patients complicated by epilepsy. They reported that if there has been no history of seizures, the finding fo a normal electroencephalogram gives more assurance that clinically evident seizures will not occur, the chances being approximately eight to one in favour of their non-appearance.

One year later, Gauger (1951) recorded EEG in 88 cerebral palsied patients and seconded the view of Aird and Cohen (above) that greater the severity of clinical involvement, more marked is the dysrrhythmia in EEG. About 80% of his patients had abnormal EEG- tracings.

Skatvedt, M. (1955) observed abnormal electro-encephalograms in 59% patients of cerebral palsy. He recorded epileptogenic discharges in 38% patients while only 20.6% had clinical epilepsy.

In a massive study over 1500 patients, Perlstein et al. (1955) noted abnormal EEG, in 90%

patients with epilepsy and in 44% patients without epilepsy, however general character of EEG in both the groups was much the same. They recorded maximum EEG abnormalities in spastics, excluding paraplegics. About 44% of their patients had seizure discharges although they were not having clinical epilepsy.The most frequent seizure discharge in their patients was of petit mal type (45%), followed by spikes (30%). In theasymmetric forms (hemiplegia etc.) they observed either unilateral on predominantly unilateral EEG abnormalities. 66% patients of right hemiplegia had left sided abnormalities while 64% of left hemiplegia had right sided defects. In the asymmetrical recordings the common abnormalities were absence or great reduction in the amount of spindle activity, absence or great reduction of parietal humps in one hemisphere and alteration of normal frequencies from side to side.

Still later in 1963 Gibbs and Gibbs reported various EEG abnormalities in these patients viz- Hypsarrhythmia, Bilateral multifocal spikes, unilateral wide spread spikes and localised spikes, 3 per sec. spikes, 14 and 6 per sec. positive spikes, irregular diffuse spikes and slow waves. Out of these, multifocal

bilateral spikes constituted commonest abnormality in 0-1 year age group. Occipital spikes were also very common while temporal spikes and petit mal type discharges were rare. They also recorded asymmetry in EEG in patients with asymmetrical clinical involvement. Slow-waves with or without spikes are common in hemiplegics (32%) thus suggesting localised cortical damage. They observed lateralizing EEG in 47% of hemiplegics and in 97% of their, lateralization was correct. Suppression of voltage production was a reliable localizing sign in EEG according to them.

Same year in 1963, Gibbs and Gibbs performed aninteresting study to predict epilepsy in cerebral palsy with help of EEG. While doing follow-up of patients with negative spiks but no epilepsy (below 2 years) they noted that during 2-9 years of age 56% of them had developed seizures. They observed no seizure in follow up of the patients having normal EEG after the age of 5 years.

Later Bauer, H. (1978) recorded 61.3% abnormal EEGs in cerebral palsy. Highest incidence of EEG abnormality was in atonic diparesis (71%). Among all the diplegics,

EEG abnormality was more frequent if mental retardation was associated (56.4%) than in the patients with normal I.Q. (29.1%). They reported that majority had focal or multifocal defects (58%) in EEG while remaining patients either had generalized changes (22.3%) or unclassifiable and boderline tracings (19.7%).

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MATERIAL AND METHODS

MATERIAL AND METHODS

Fifty patients of various types of cerebral palsy, attending the out patient department of pediatrics or admitted in pediatric ward, of Maharani Laxmi Bai Medical College, Jhansi, from September 1987 to August 1988, were included in this study. Most of the patients who were admitted, had some associated complications notably epilepsy.

METHODS

Detailed history pertaining to perinatal events was recorded from the mother of every patient. Regarding Antenatal history questions related to following events were asked-like consanguinity, infections, anemia, toxemia of pregnancy, hypertension, diabetes, malnutrition, any chronic systemic illness, cervical incompetance, polyhydramnios/oligohydramnios, multiple-pregnancy, smoking, irradiation, drug intake, elderly or teenage mother, socio-economic status, unwed-mothers, Bleeding P/V and premature rupture of membranes. The natal history comprised of-duration of labour, mode of delivery, birth trauma, respiratory distress, breech delivery, and precipitate delivery.

The postnatal history included history of-Active resuscitation required, Apgar score, birth-weight, gestational-age, cyanosis, Listlessness, hypoxia, Convulsions, Jaundice, Septicemia, hypoglycemia, Blood-Group incompatibilities, congenital anomalies and hospitalization any time during neonatal period.

History of neurological problems or any significant systemic illness during infancy and childhood, upto the age of 5 years, was then asked-viz. Meningitis, Encephalitis, cerebrovascular accidents or encephalopathy.

History of epilepsy was obtained in full detail from the mother of the patient paying particular emphasis to age of onset, frequency of seizures, duration of each episode, precipitating factors like fatigue, light, emotional-upset; type of seizure, pre and post convulsive events and family history of epilepsy. The nature and duration of any anti convulsant drug intake was also recorded.

Developmental history was recorded in all the spheres. Gross- motor, fine motor, social and speech mile stones attained till date were recorded in every case. In all four developmental fields, Quotient was calculated by dividing the developmental age from chronological age and then multiplying this value by 100. Now by taking the mean of this quotient in all four developmental fields, the developmental-quotient was calculated (Prabhakar and Kumar, 1983).

Every patient was examined in detail, paying particular emphasis to the extent of functional and anatomical neurological deficit; abnormality of gait or posture or movement. Any Abnormality in the size and shape of the head was also looked for. The head circumference was measured at the level of external occipital protuberance posteriorly and supra orbital ridges anteriorly. The patient was also examined for any cerebellar sign and sensory deficit. Every patient was specifically asked for the associated handicaps viz. seizures, visual defects, speech problems, hearing defects, communication and emotional defects, learning problems, mental retardation or any congenital abnormality.

Fundus examination and X-ray skull, both Anterio-posterior and lateral views, were performed in every case.

The electroencephalographic recording was done in every patient, using eight channel EEG recorder of Medicare, Chandigarh. Every patient was sedated before hand, using syrup chloral hydrate in dose of 25-50mg/kg. Ten twenty system of electrode plasscement, accepted internationally, was used. Electrodes were fixed in position using Bentonite paste. Both monopolar and Bipolar montages were recorded in every case. Only routine recording was done in every patient as potentiation was not feasible due to obvious reasons. About half an hour long record was taken in every case. Every record was studied as per internationally accepted criteria given by Kiloh et al, 1982.

In the end, findings were tabulated and data were analysed statistically.

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OBSERVATIONS

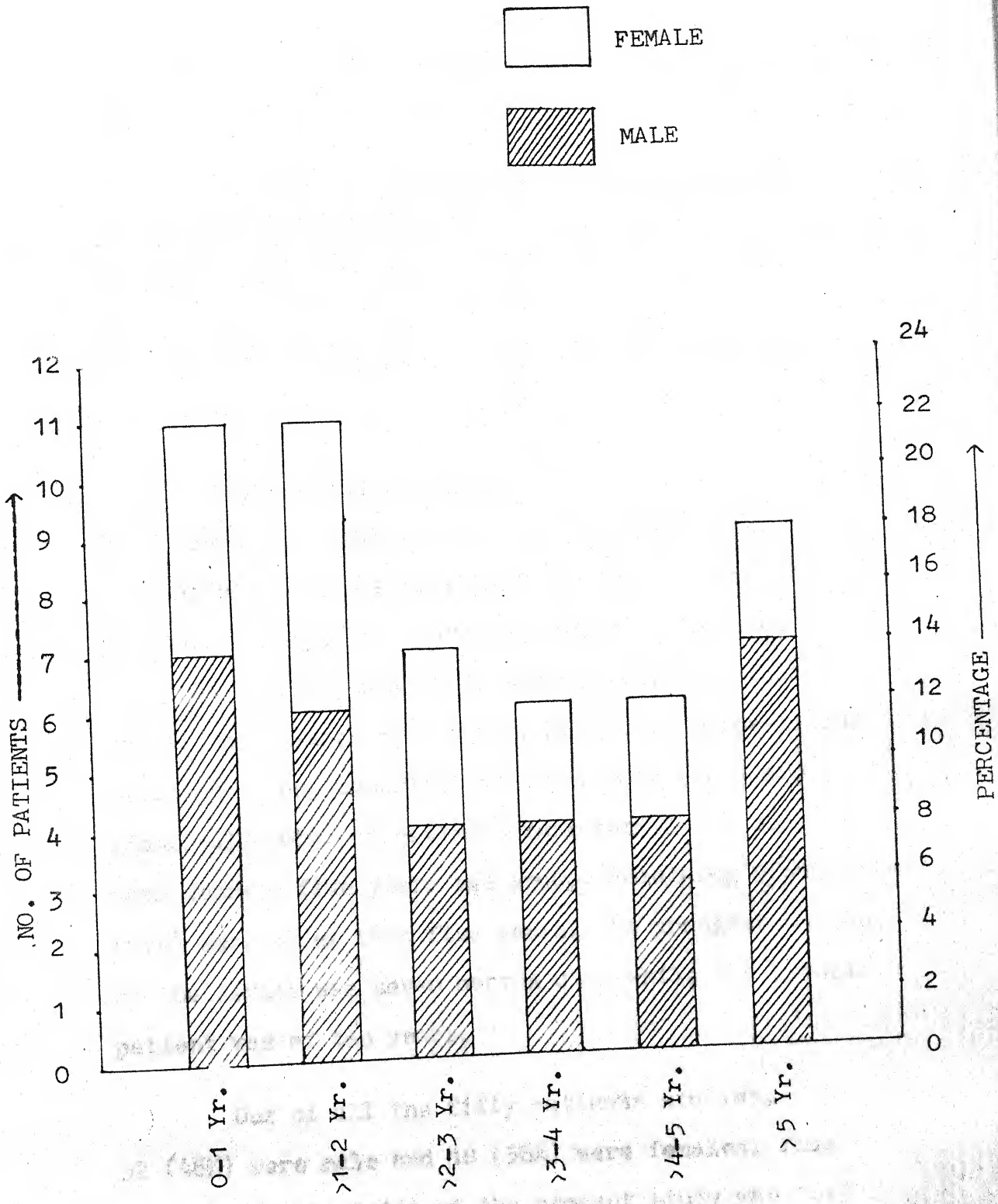

FIG. 1 - AGE AND SEX DISTRIBUTION

OBSERVATIONS

The present study entitled "Electro encephalography and Epilepsy in cerebral - Palsy" was perofrmed over fifty patients. Electro - clinical features of various types of cerebral Palsy were studied and following results were observed:-

The age distribution of the patients under study is shown in Fig. 1.11 patients (22%) were upto one year of age while similar number of patients belonged to, more than one-to-two years age group. 7 patients (14%) were in the more than two-to-three years of age bar. More than three-tofour years age group comprised of 6 patients (12%) and identical number of patients represented the more than four-to-five years age group. Remaining 9 patients (18%) were older than five years. The youngest patient of the series was seven months old, while the oldest patient was of ten years.

Out of all the fifty patients studied, 32 (46%) were male and 18 (36%) were females. Thus the male female ratio of the present study was 16:9

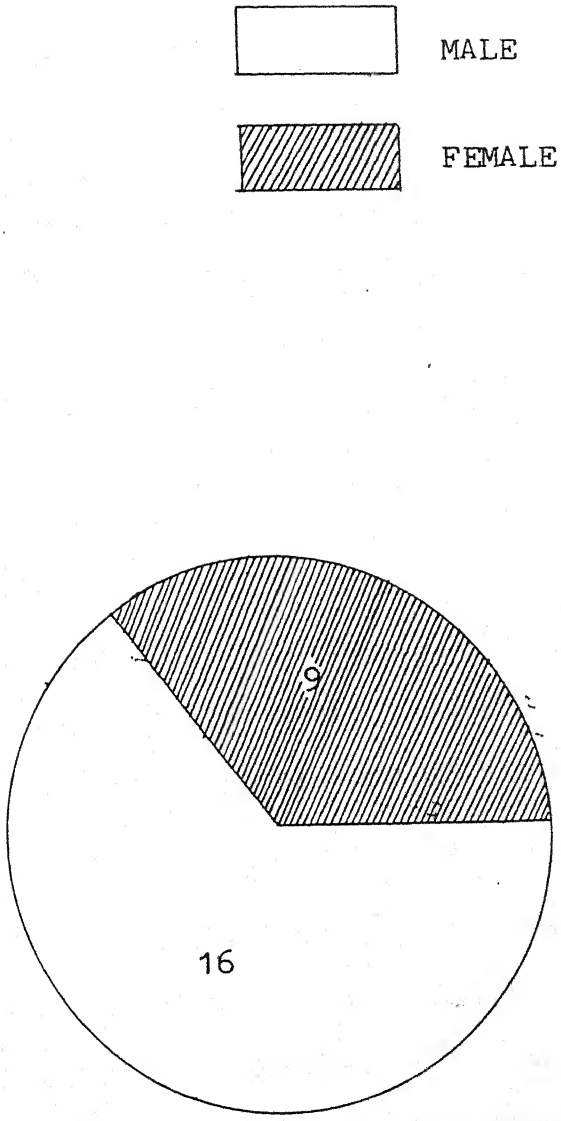

FIG. 2 MALE : FEMALE RATIO

(Fig.2). The over all sex distribution in different age groups is also shown in Fig.1.

Among all the fifty patients, 22 (44%) gave history suggestive of birth asphyxia in the form of-delayed Cry, Cyanosis and listlessness. Distribution of all the patients according to etiology is mentioned in table 1, out of all the patients with history of birth asphyxia some revealed history of other associated factors also, like Septicemia (4%), prematurity (8%) and small for date babies (6%). Remaining 26% patients had history of birth asphyxia only.

In all, 15 (33.3%) patients were born prematurely and out of them 4 had associated birth asphyxia also. Most of the babies (70%) born prematurely were below 30-32 weeks of gestational age as stated by the mother or mentioned in hospital discharge records. The exact birth weight of the babies could not be ascertained as mothers did not remember it.

TABLE 1 : ETIOLOGICAL - DISTRIBUTION

Etiology	Number of Patients	Percentage
A. Birth Asphyxia	22	44
-With Prematurity	4	8
-Small for date Babies	3	6
-With Septicemia	2	4
B. Prematurity	11	22
C. Septicemia-Hypoglycomia	3	6
D. Post-Encephalitic	5	10
E. Post-Meningitic	3	6
F. Post-Icteric	2	4
G. Unknown-Cause	4	8
TOTAL	50	100

3 patients (6%) revealed history suggestive of septicemia and hypoglycemia. All of them elaborated history of delayed onset of feeding and were hospitalized then. 5 patients (10%) had history suggestive of postencephalic onset. All of them were hospitalized though only three displayed old records.

2 patients (4%) gave history of severe and prolonged jaundice, during early postnatal period. Both were hospitalized during their ailment and one had undergone exchange transfusion while other could not, though his discharge records revealed serum bilirubin level of 43 mg% at that time. None had any major blood-group incompatibility.

Post Meningitic etiology could be discovered in 3 patients (6%) of the present study. Out of them, 1 patient had septic meningitis four months back and other two patients had tubercular Meningitis. All were hospitalized during their ailment.

Etiology of 4 patients (8%) could not be ascertained inspite of detailed antenatal, natal and postnatal history. The period of infancy and childhood, upto the age of five years, was also uneventful, in them.

The distribution of patients according to clinical types of cerebral Palsy is shown in Table 2.

Most common type of motor disorder was spasticity, showing signs of pyramidal tract involvement, as 38 (76%) patients belonged to this type only.

TABLE 2 : CLINICAL TYPES OF CEREBRAL PALSY

Clinical Types of Cerebral Palsy Motor Disorder	Topographic Distribution	Number of Patients		Percentage	
A. SPASTICITY	1.Monoparesis	1	38	2	76
	2.Diparesis	23		46	
	3.Triparesis	1		2	
	4.Right Hemiparesis	5		10	
	5.Left Hemiparesis	3		6	
	6.Quadriparesis	5		10	
B. HYPOTONIA	1.Diparesis	5	7	10	14
	2.Quadriparesis	2		4	
C. ATHETOSIS	Quadriparesis		1		2
D. ATAXIA	Quadriparesis		1		2
E. MIXED (Rigidity & Tremors)	Quadriparesis		3		6
TOTAL			50		100

Among all the spastics, diplegic distribution was seen in 23 (46% patients. Next common disturbance in spastic patients was hemiparesis, in which 5 (10%) had right sided

and remaining 3 (6%) had left sided affection. 5 (10%) patients had spastic Quadriparesis while, 1 (2%) each belonged to Monoparesis and Triparesis types. The patient with monoparesis had upper motor neurone signs in left lower limb only while triparetic patient had involvement of both lower limbs alongwith right upper limb.

7 (14%) cases belonged to hypotonic group. They all had hypotonia along with hyper-reflexia and extensor plantar response. 5 (10%) had diplegic affection while remaining 2 (4%) had Quadriplegic involvement.

Only 1 (2%) patient in this study showed atheloid movements involving all four limbs and another 1 (2%) patient had ataxia of both the upper and lower limbs. Remaining 3 (6%) patient were categorized as mixed cerebral Palsy as they all had rigidity and tremors both in all the four limbs.

According to topographic distribution the commonest type was diparesis which was seen in

28 (56%) patients and of these 23 belonged to spastic type and rest 5 had hypotonia.

Commonest associated anomaly was microcephaly. 62% patients of this series had microcephaly as their head circumference was below 3rd percentile of Harvard standard. Distribution of microcephaly in various clinical types of cerebral palsy is mentioned in Table 3.

TABLE 3 : DISTRIBUTION OF MICROCEPHALY IN CEREBRAL PALSY

Clinical Types of Cerebral Palsy		Microcephaly		
Motor Disorder	Topographic Distribution	Number of Patients		Percentage
A. SPASTICITY	1. Monoparesis	-	21	67.7
	2. Diparesis	15		
	3. Triparesis	-		
	4. Right Hemiparesis	1		
	5. Left Hemiparesis	1		
	6. Quadriparesis	4		
B. HYPOTONIA	1. Diparesis	5	7	22.6
	2. Quadriparesis	2		
C. ATHETOSIS	Quadriparesis		1	3.2
D. ATAXIA	Quadriparesis		-	-
E. MIXED	Quadriparesis		2	6.5
TOTAL			31	100

Out of all the patients of cerebral palsy with microcephaly, 21 (67.7%) belonged to spastic group, 7 (22.6%) belonged to hypotonic group, 1 (3.2%) belonged to athetosis and rest 2 (6.5%) were of mixed type. No patient of ataxic type had microcephaly.

The occurance of microcephaly was highest in hypotonic group of cerebral palsy where all the seven patients (100%) had microcephaly. The sole patient of athetosis also had microcephaly. Incidence of microcephaly among spastics was 55.2% as 21 out of 38 spastics had microcephaly. No patient of ataxia had microcephaly, but 2 out of 3 (66.6%) patients of mixed cerebral palsy had microcephaly. (Table 3).

Among other significant congenital anomalies, it is worth-while to mention that one patient had patent-ductus-arteriosus and had undergone surgery for the same at one year of age. He was suffering from spastic diparesis. Another patient had bilateral cataract, microcephaly, seizures, and hepatomegaly and thus was diagnosed to be a case of congenital Rubella syndrome. He was also low birth weight at birth. He also

had spastic diparesis. One patient who belonged to spastic quadriparesis group had associated arthrogryposis multiplex congenita.

History of seizures was present in 28 (56%) patients. Distribution of sezures in various types of cerebral palsy is depicted in Table 4. Majority of patients belonged to spastic group (75%) and quiet a sizeable number of hypotonic patients (21.3%) also had seizures. Only 1 (3.7%) patient of mixed type comprised the epleptics. It was observed that out of all the spasties 55.3% had epilepsy, and out of all hypotonic patients 85.7% had epilepsy, while none of the ataxic or athetoid patient had any seizure. In the mixed type 1 out of 3 patients (33.3%) had epilepsy.

Majority (60%) of epileptics had several convulsions daily.

Generalised tonic-clonic was the commonest clinical type of seizure observed. It was seen in 12 out of all 28 epileptic patients (42.9%). The frequency

TABLE 4 : DISTRIBUTION OF EPILEPSY IN CEREBRAL PALSY

Clinical types of Cerebral Palsy		Seizure Present			Types of Seizurs						
Motor Disorder	Topographic Distribution	Number of patients		%	Gen. Tonic clonic	Myo clonic	Gen. Tonic	Focal	Psy-cho mot-or	Mix-ed	Gen. ton-ic
A. SPASTICITY	1.Monoparesis	1	21	75	-	-	-	1	-	-	-
	2.Diparesis	11			5	2	2	-	1	1	-
	3.Triparesis	-			-	-	-	-	-	-	-
	4.Right Hemiparesis	4			3	-	-	1	-	-	-
	5.Left Hemiparesis	-			-	-	-	-	-	-	-
	6.Quadriparesis	5			2	-	1	-	-	1	1
B. HYPOTONIA	1.Diparesis	5	6	21.6	1	3	-	1	-	-	-
	2.Quadriparesis	1			1	-	-	-	-	-	-
C. ATHETOSIS	Quadriparesis		—	—	-	-	-	-	-	-	-
D. ATAXIA	Quadriparesis		—	—	-	-	-	-	-	-	-
E. MIXED	Quadriparesis		1	3.7	-	-	-	-	1	-	-
TOTAL			28	100	12	5	3	3	2	2	1
					42.9%	18%	10.7%	10.7%	7.1%	7.1%	3.5%

distribution of various clinical types of epilepsies is also shown in table 4. Among all the patients with generalised tonic clonic type of seizure, 5 belonged to spastic diparesis, 3 to right hemiparesis, 2 to Quadriparesis and 1 each to hypotonic diparesis and hypotonic quadriparesis.

Myoclonic seizure was the second next common type, observed in 5 (18.0%) patients. All were diparetics; 3 were hypotonic and remaining 2 were spastic. All of these patients had, had several episodes daily.

Next in the sequence was generalised tonic epilepsy and focal epilepsy as both were seen in 3 (10.7%) patients each. 2 of the spastic diparetic and 1 of the spastic quadriparetic had generalised tonic epilepsy while one patient each, belonging to spastic monoparesis, spastic right hemiparesis and hypotonic diparesis types were labelled as focal and jacksonian epilepsy.

2 (10.7%) patients each had psychomotor and mixed type of epilepsy, out of two patients with psychomotor epilepsy one had spastic diparesis

and other-one had mixed type of cerebral palsy. Both presented as episodic abnormality of behaviour. 2 patients had more than one type of epilepsy and thus labelled mixed epilepsy. Out of these 2, one had spastic diparesis and other had spastic Quadriparesis.

Remaining 1 patient (3.5%) had generalised clonic seizures and belonged to spastic quadriparesis. Fundus examination was performed in all the fifty patients under study, where two patients of post meningitic onset revealed bilateral optic atrophy. X-Ray skull did not reveal any abnormality in any of the cases.

The average developmental quotient (D.Q.) among all the fifty patients of cerebral palsy, was 34.9%. Differential developmental quotient in different types of cerebral palsy is shown in Table 5.

Maximum developmental retardation was seen in Athetosis and spastic Triparesis, where developmental quotients of only 11% and 19% were observed respectively. On the other hand least retardation of development was seen in spastic left hemiparesis, spastic monoparesis

TABLE 5 : DEVELOPMENTAL QUOTIENT IN VARIOUS TYPES OF CEREBRAL PALSY

Clinical type of Cerebral Palsy Motor Disorder	Topographic Distribution	Developmental Quotient(D.Q.)	Mean (D.Q.)
A. SPASTICITY	1.Monoparesis	57.0%	36.9%
	2.Diparesis	33.3%	
	3.Triparesis	19.0%	
	4.Right Hemiparesis	49.6%	
	5.Left Hemiparesis	59.7%	
	6.Qudriparesis	26.6%	
B. HYPOTONIA	1.Diparesis	27.0%	25.14%
	2.Quadriparesis	20.50%	
C. ATHETOSIS	Quadriparesis	11.0%	
D. ATAXIA	Quadriparesis	65.0%	
E. MIXED	Quadriparesis	30.3%	
Average			34.9%

and ataxic types. Overall mean D.Q. in spastics was 36.9% and in hypotonic patients was 25.14% while in mixed cerebral palsy the mean value was 30.3%.

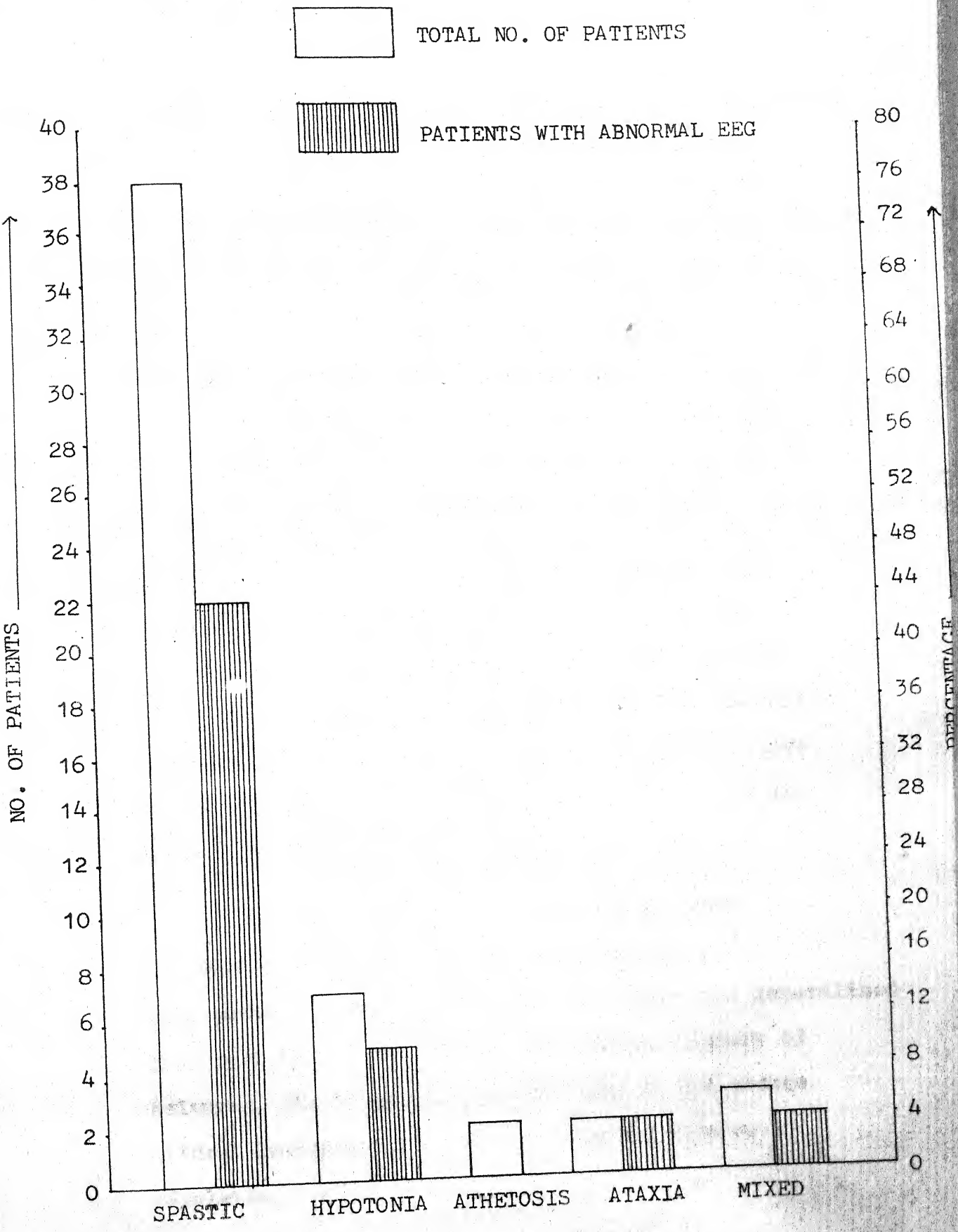

FIG. 3 - PATIENTS WITH ABNORMAL ELECTROENCEPHALOGRAMS

The mean developmental Quotient in spastic diparesis was 33.3% and in hypotonic diparesis, the corresponding value was 27.0%. Out of all the fifty patients under study 29 (58%) patients had developmental quotient values below the overall average value of 34.9%. The mean value of developmental quotient in all the patients suffering from epilepsy was 30.75% while the corresponding average value in patients having microcephally was 28.97%.

Out of all the 50 patients studied in the present series, 30 (60%) patients had one or the other abnormality in electro-encephalography. Rest 20 (40%) patients had essentially normal EEG records. The details of electroencephalographic abnormalities in various types of cerebral palsy are mentioned in table 6,7 and Fig.3.

Out of 38 spastic patients, 22 patients (57.9%) had abnormal electroencephalograpms. The sole patient of monoparesis had both focal and generalised abnormalities in EEG-record, he also had history of seizures. The focal changes were seen in the contra lateral hemisphere in the form of spike slow wave complexes.

TABLE 6 : ABNORMAL ELECTROENCEPHALOGRAMS IN VARIOUS TYPES OF CEREBRAL PALSY

Clinical types of Cerebral Palsy		Total number of patients		Abnormal Electroencephalograms							
Motor Disorder	Topographic Distribution			Total		Generalised Abnormality		Focal Abnormality		Focal and Generalised Abnormality	
A. SPASTICITY	1.Monoparesis	1		1		-		1		-	
	2.Diparesis	23		13		6		2		5	
	3.Triparesis	1		-		-		-		-	
	4.Right Hemiparesis	5	38	5	22	-	7	3	8	2	7
	5.Left Hemiparesis	3		1		-		1		-	
	6.Quadriparesis	5		2		1		1		-	
B. HYPOTONIA	1.Diparesis	5	7	3	5	2	3	-		1	2
	2.Quadriparesis	2		2		1		-		1	
C. ATHETOSIS	Quadriparesis	1		-		-		-		-	
D. ATAXIA	Quadriparesis	1		1		1		-		-	
E. MIXED	Quadriparesis	3		2		2		-		-	
TOTAL		50		30		13		8		9	

TABLE 7 : LATERALITY OF ELECTROENCEPHALOGRAPHIC ABNORMALITIES

Clinical types of Cerebral Palsy Motor Disorder	Topographic Distribution	Right (%)	Bilateral (%)	Left (%)	Total (%)
A. SPASTICITY	1. Monoparesis (Left Crural)	100	-	-	100
	2. Diparesis	15.4%	84.6%	-	100%
	3. Triparesis (Both lower limbs and Right upper limb)	-	-	-	-
	4. Right Hemiparesis	-	33.4	66.6	100
	5. Left Hemiparesis	100	-	-	100
	6. Quadriparesis	-	50%	50%	100
B. HYPOTONIA	1. Diparesis	-	100	-	100
	2. Quadriparesis	-	100	-	100
C. ATHETOSIS	Quadriparesis	-	-	-	-
D. ATAXIA	Quadriparesis	-	100	-	100
E. MIXED	Quadriparesis	-	100	-	100

Out of all 23 patients of spastic diparesis, 13 (56.5%) had abnormal EEG records. Most of them (11 patients) had generalised changes though 5 had additional focal defects. 2 patients had only focal changes. Sole patient of triparesis had normal recording.

Interesting changes in electroencephalograms were observed in spastic hemiparetic patients. In the present series, 5 patients had right sided affection and all revealed changes in centralateral hemishpere. All had focal abnormalities though two of them also had generalised changes. Similarly, 1 out of 3 patients with left spastic hemiparesis, had abnormal EEG and he also showed focal changes in contralateral side.

Two patients of spastic quadriparesis had essentially defective tracings and one had generalised while other had focal defects. Other details of abnormal electroencephalograms are depicted in tables 6 and 7.

It is important to mention here that out of all 30 patients with abnormal tracings only 20 gave history of epilepsy while 10 patients never experienced seizures. Retrospectively the occurance of EEG abnormalities

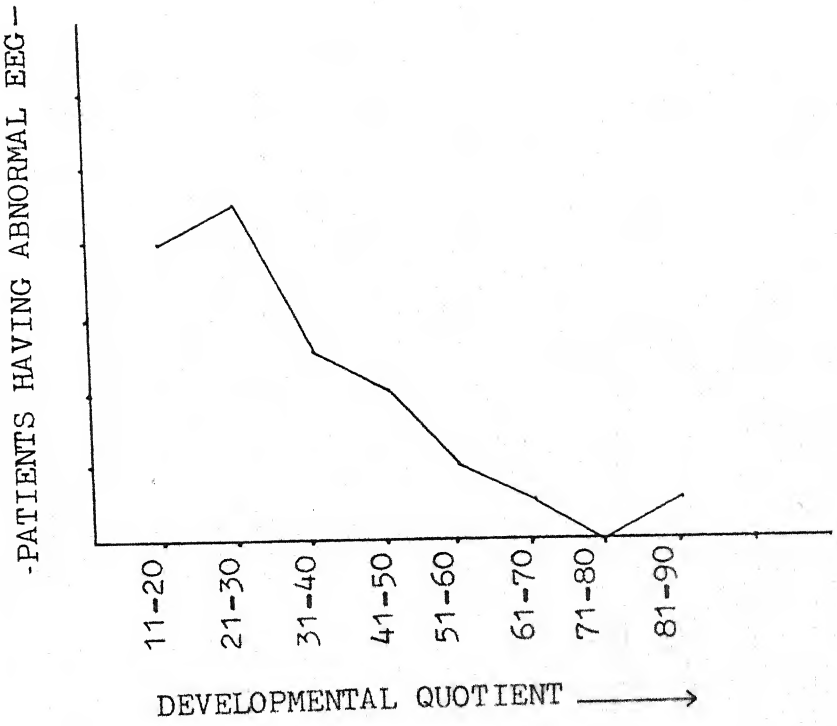

FIG. 4 - EEG ABNORMALITY AND DEVELOPMENTAL QUOTIENT

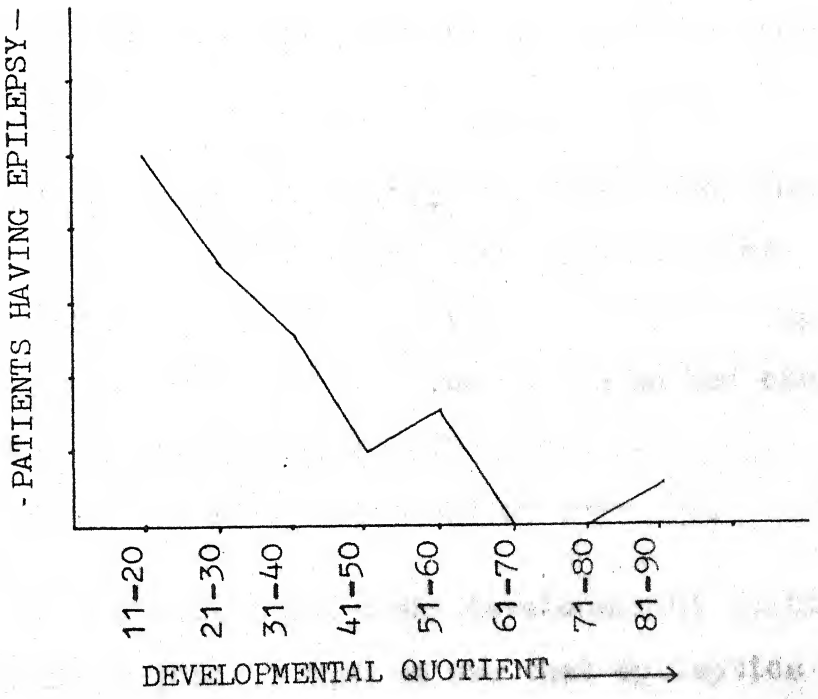

FIG.5 - EPILEPSY AND DEVELOPMENTAL QUOTIENT

in epileptics was 67.8% as out of 28 epileptics in the series, 19 patients had abnormal electroencephalograms. The chances of EEG abnormality were maximum in myoclonic seizures as all of them (100%) had abnormal tracings.

Among all the patients with abnormal EEG tracings, the mean developmental quotient was 32.7% while the overall mean of all the patients was 34.9%. Patients having abnormal electroencephalograms, in different grades of developmental quotient are depicted in Fig. 4.

It is clear from this figure that lower the developmental quotient, higher are the chances of EEG-abnormalities. The sole patient in 81-90% group had spastic hemiparesis (Rt.) and in EEG he had changes suggestive of left hemispherical damage. None had developmental quotient, below 10% or over 90%.

If epileptics and their developmental quotients are correlated (Fig.5) then we see that epileptics have more chances of developmental retardation. Majority (85%) of epileptics had developmental quotient below 50%. The patient belonging to 81-90% group had convulsions associated with septic meningitis, when he had it four months back.

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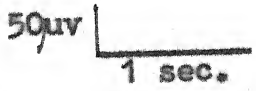
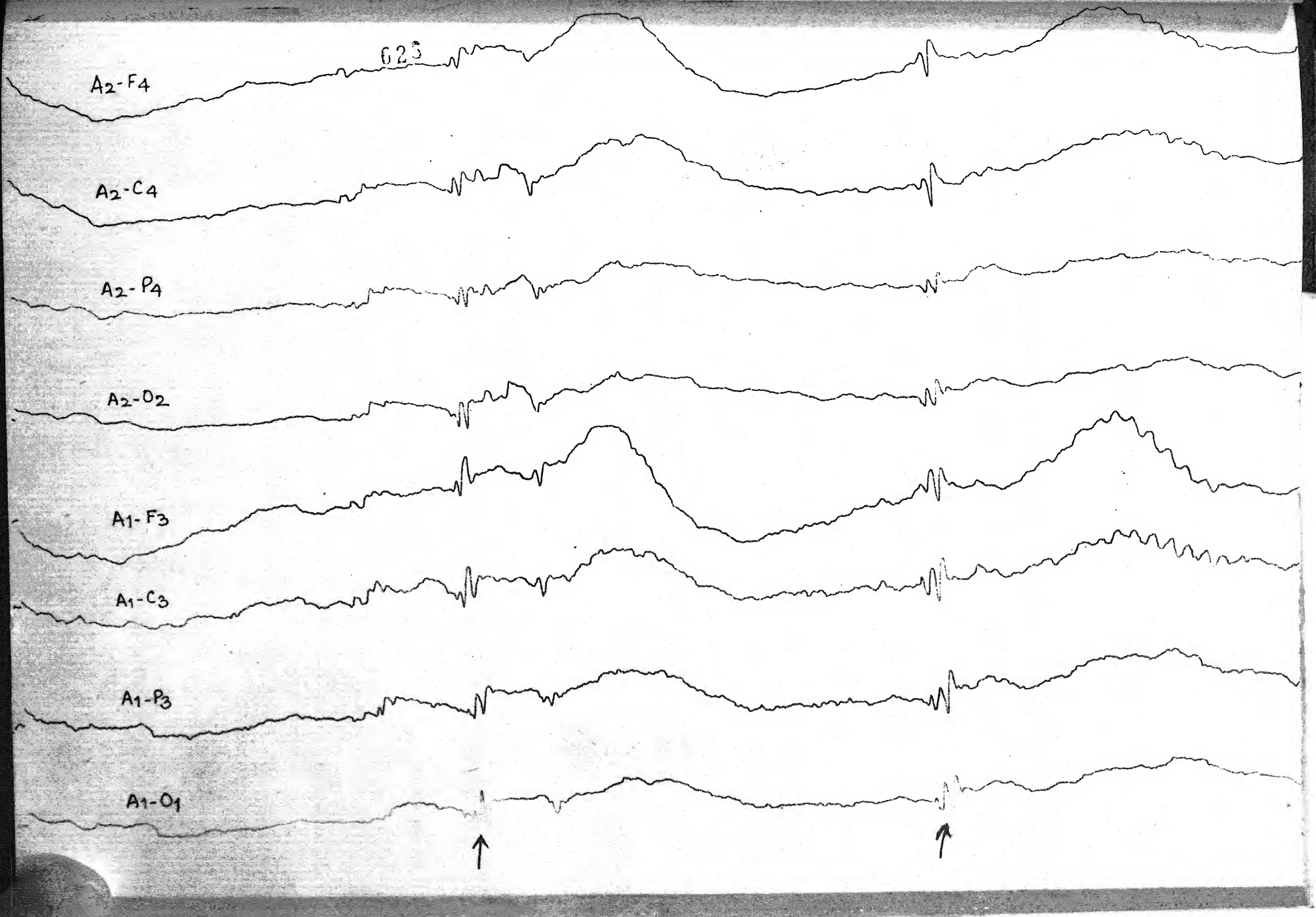
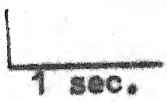
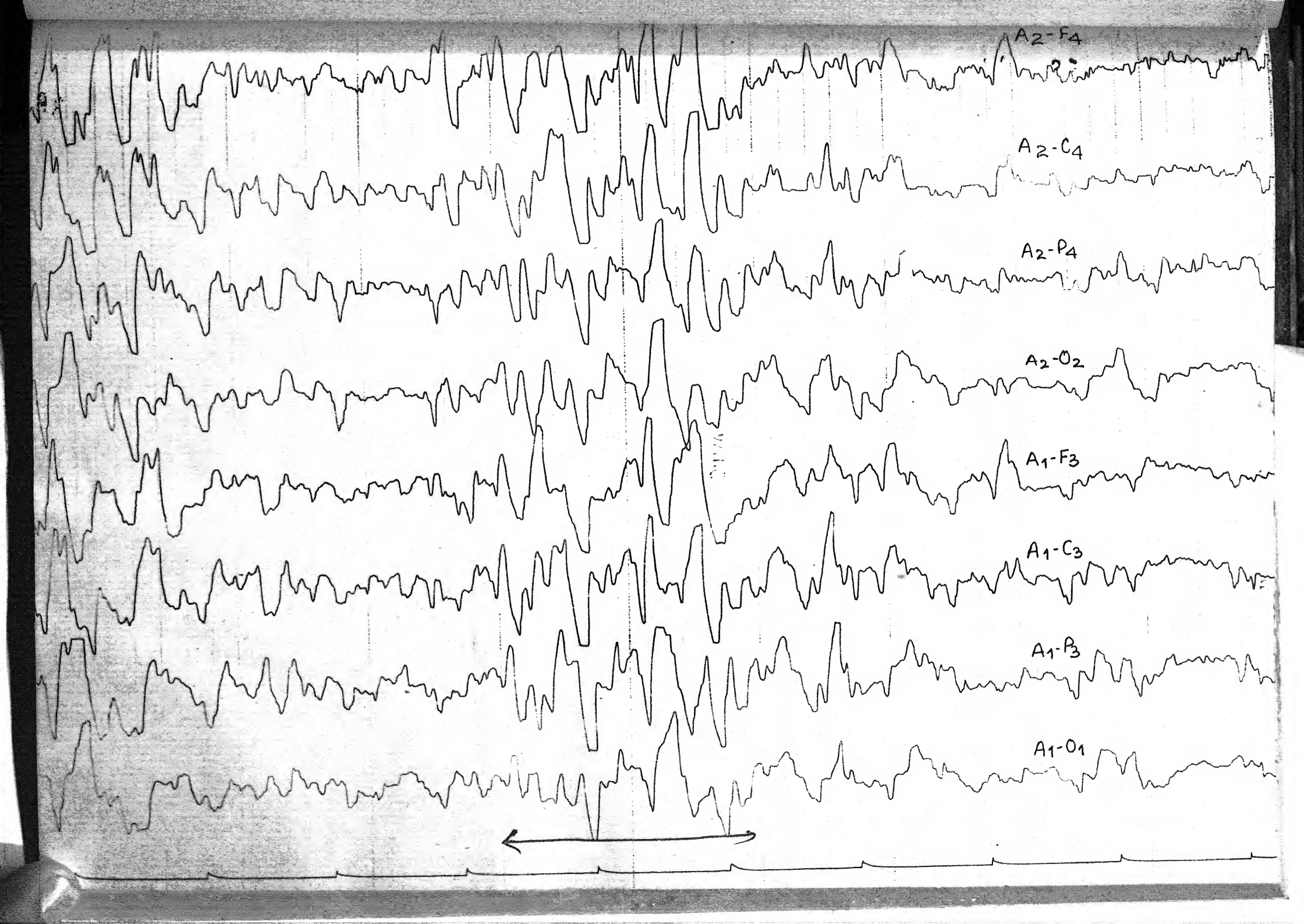
EEG No. 1 - Generalised and synchronous spikes, suggesting generalised epilepsy.



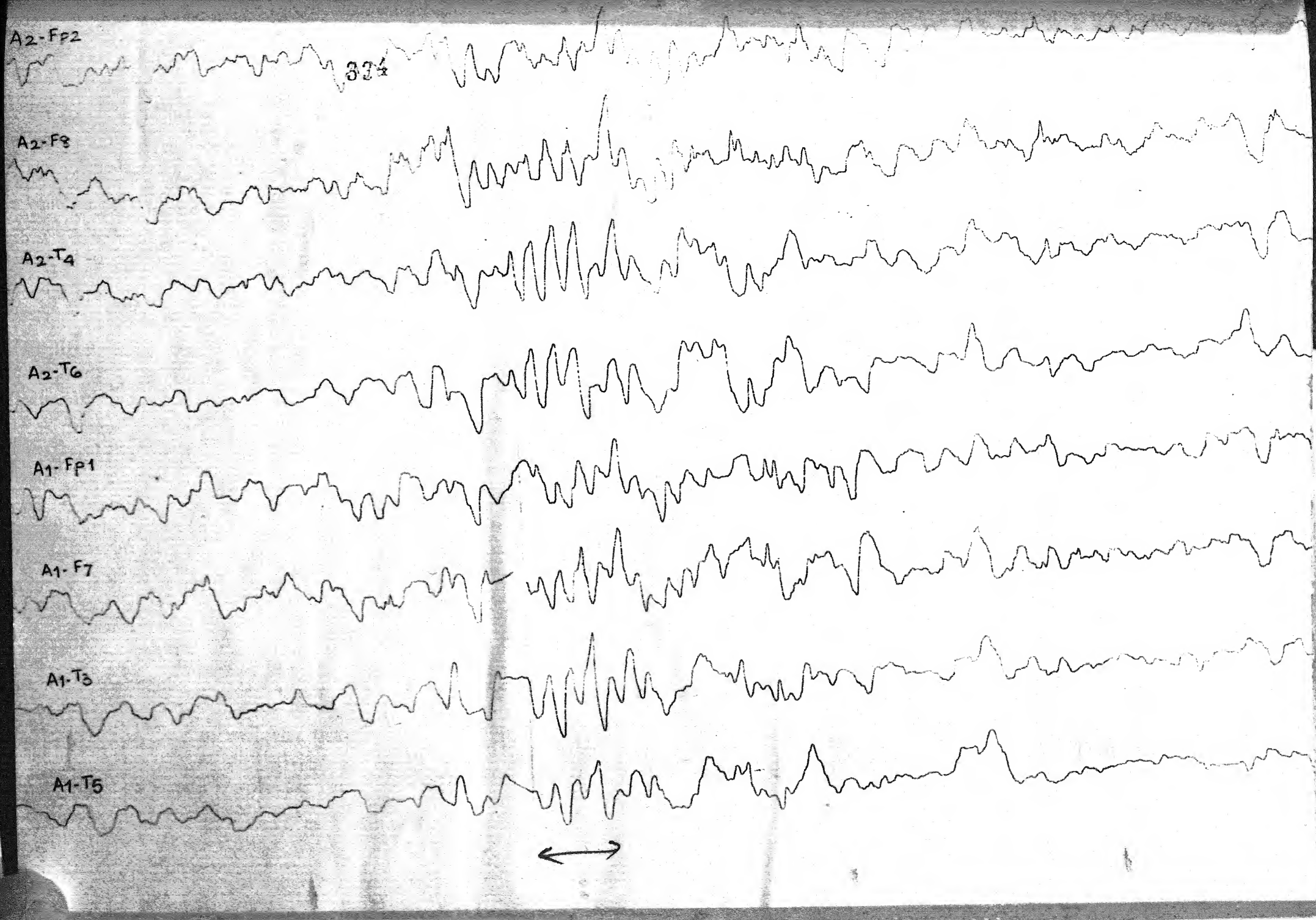

625
A2-F4
A2-C4
A2-P4
A2-O2
A1-F3
A1-C3
A1-P3
A1-O1

EEG No. 6 - Burst of slow-and-sharp waves, suggesting generalised epilepsy

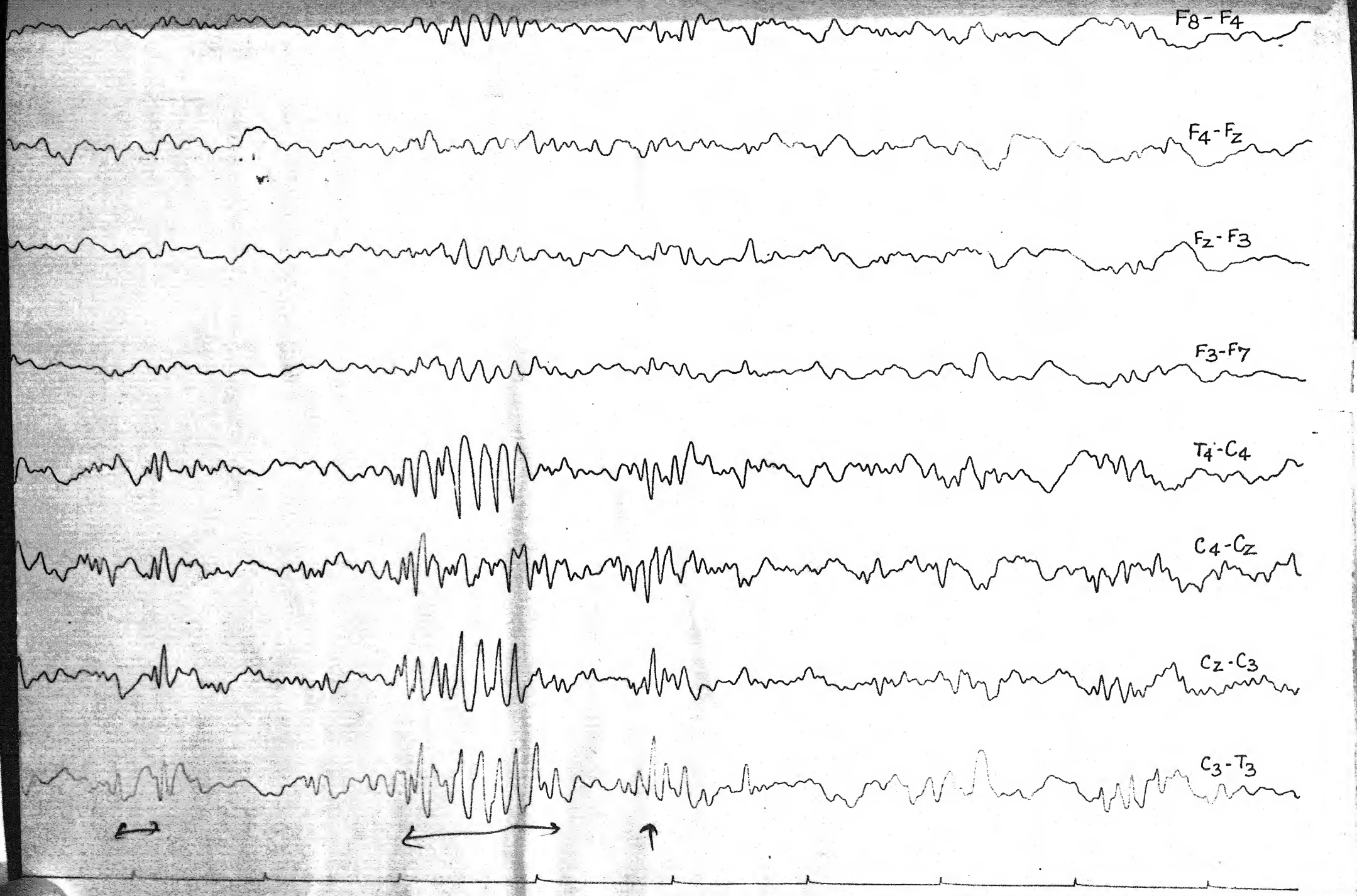



A2-F4
A2-C4
A2-P4
A2-O2
A1-F3
A1-C3
A1-P3
A1-O1

EEG No. 10 - Intermittent slowing suggestive of generalized brain damage.

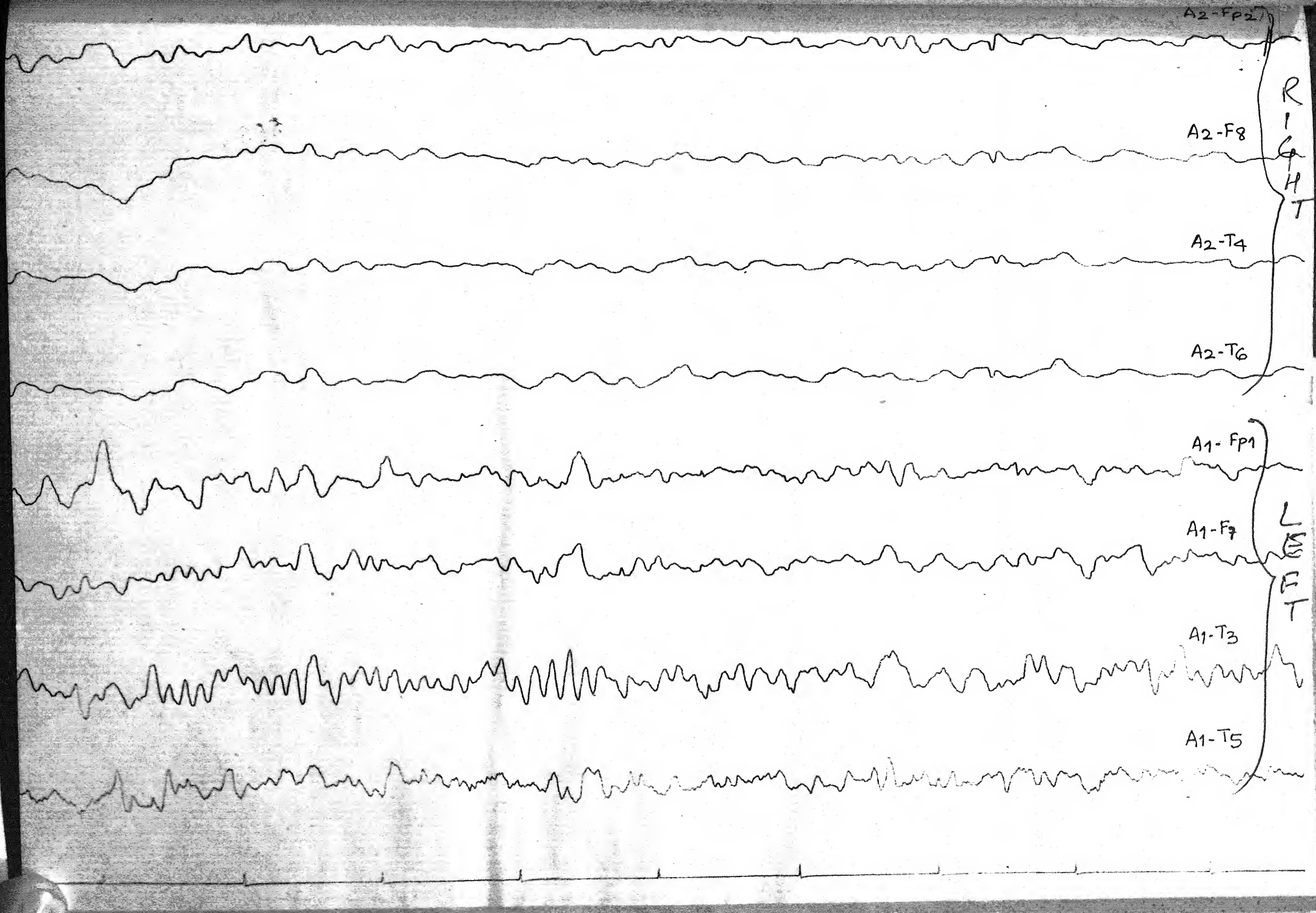

A2-Fp2
334
A2-F8
A2-T4
A2-T6
A1-Fp1
A1-F7
A1-T3
A1-T5

EEG No.18 - Burst of spikes and sharp waves and phase reversals, suggesting some focus around left postero central (C_z) area.


F8 - F4
F4 - Fz
Fz - F3
F3 - F7
T4 - C4
C4 - Cz
Cz - C3
C3 - T3

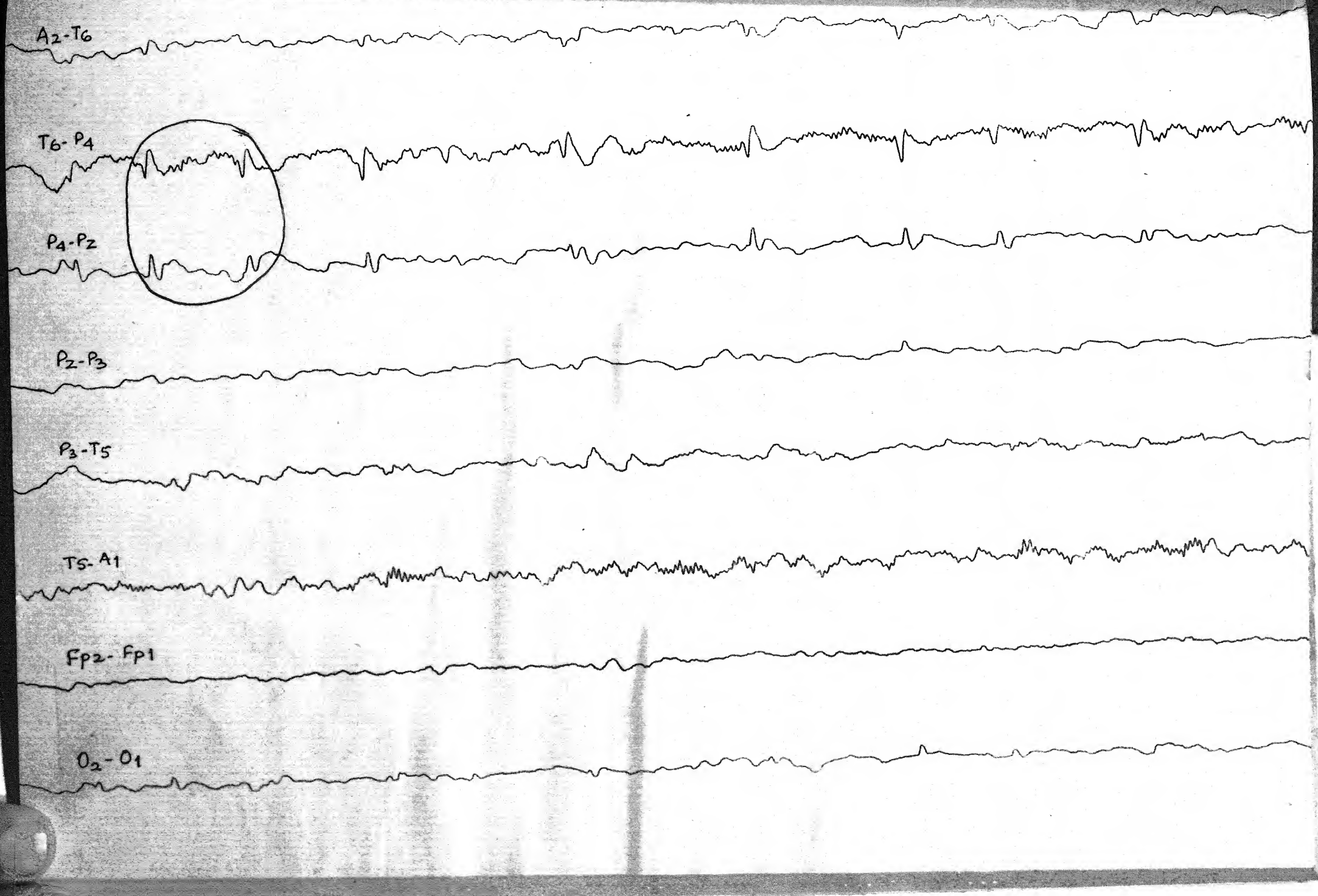
EEG No. 33 - Decreased amplitude and frequency on right side, suggestive of right hemispherical damage.

50μv
1 sec.

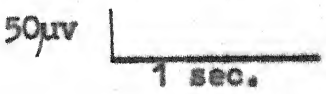

A2-Fp2
A2-F8
A2-T4
A2-T6
RIGHT
A1-Fp1
A1-F7
A1-T3
A1-T5
LEFT

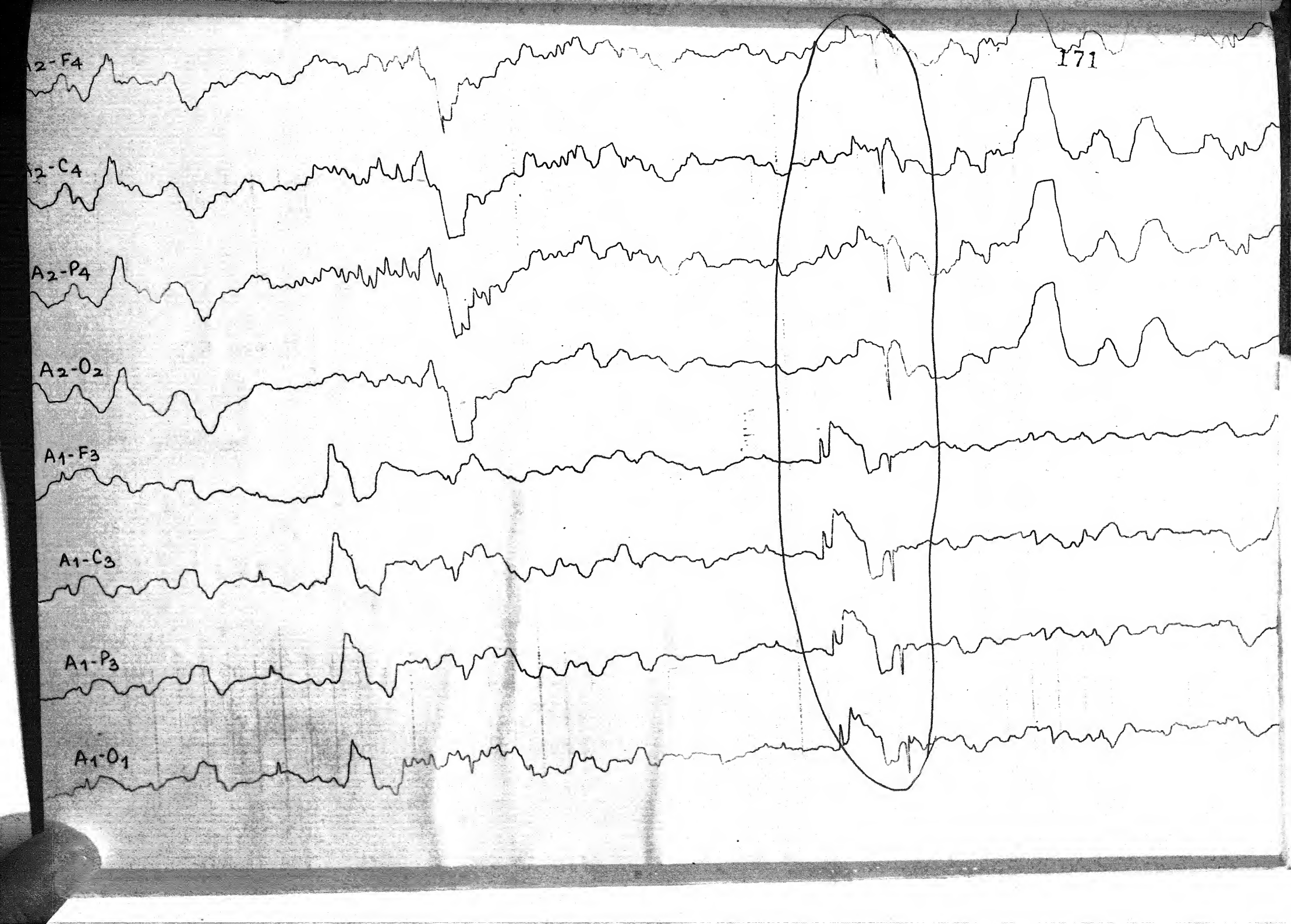
EEG No. 35 - Multiple phase reversals, suggestive of some focus around posterior parietal area (P_4)

50μv
1 sec.


A2-T6
T6-P4
P4-Pz
Pz-P3
P3-T5
T5-A1
Fp2-Fp1
O2-O1

EEG No. 37 - Generalised spikes which are synchronous also, suggestive of generalised epilepsy




A2-F4
A2-C4
A2-P4
A2-O2
A1-F3
A1-C3
A1-P3
A1-O1

DISCUSSION

DISCUSSION

The study of cerebral palsy has gained a great impetus, due to the development of specific research projects in this field. One of them is the application of electroencephalography in these patients. Electro-encephalography, in that it affords data on cerebral physiology, is of considerable significance in the conditions, associated with cerebral pathology and abnormal physiology. This is especially true in those cerebral palsied children whose condition is complicated by convulsive disorders. Considering the problem of epilepsy in cerebral palsy and newer modalities in diagnostic field, it deemed worth while to perform an electro-clinical study on these patients.

The present study was conducted on 50 patients with various presentations of cerebral palsy, who attended the out patient department of Pediatrics, M.L.B. Medical College, Jhansi, from September 1987 to August 1988.

Male dominance in the patients of cerebral palsy is a universally known fact (Pertstein et al., 1955, Garg et al., 1965, Sharma et al., 1981).

The reason for this male dominance is difficult to explain. However, Lennox (1953) attributed this predilection of males to their greater liability to congenital cerebral defects and birth injuries. We also experienced male preponderance, as male : female ratio in our study was 16:9.

Birth asphyxia was the commonest predisposing factor in our study, as 44% (22) of our patients were asphyxiated at birth. Out of these, 8% (4) patients were prematures while rest 36% (18) had suffered from birth asphyxia inspite of being full term. The reason could be poor obstetric care, home deliveries, mismanaged-delivery and paucity of diagnostic and therapeutic modalities in this field. Udani (1963) and Garg et al., (1965) reported asphyxia in 30% and 33% cases respectively. Anderson (1952) stated that, third to half of all cases of cerebral palsy had evidence of anoxia at birth. Eastman and Deleon (1955) found foetal distress to be four times commoner in cerebral palsy than in general population. Brann (1986) reported that Ulegyria and status marmoratus of basal ganglia are basic pathological changes in these asphyxiated babies. The lesions are principally in peripheral and dorsal areas of cerebral cortex, involving necrosis of gyri at the depth of sulci and the neuronal nuclei of basal-ganglia and brain-stem.

Several other workers had reported prematurity as the principal risk factor (Eastman et al., 1955, Churchill 1974, Prabhakar 1983). The average incidence of prematurity in cerebral palsy, according to them is around 35%, thus prematurity is five times more common in cerebral palsy than general population. About one third (30%) of our patients too were prematures though 8% of them had suffered from birth asphyxia also. Prematurity principally causes subependymal-intraventricular hemorrhage. These lesions are located at the centre of hemisphere in germinal matrix along ventricular region, with sparing of cortical mantle (Brann, 1986). Deep white matter of periventricular region at the angles of ventricles, may undergo cystic degeneration -Peri-ventricular Leukomalacia.

The reported incidence of cerebral palsy due to hyperbilirubinemia is highly variable. This disparity could be due to the size of sample under study and occurance of Rh (D) negative individuals in that community. The reported values range from 17.5% (Brandt et al., 1958) to 5.8% (Martin 1960). We found that 2 (4%) cases in our study had severe jaundice during postnatal period one developed athetosis and other one developed spastic diparesis. Garg et al. (1965)

could reveal history of jaundice in 14.5% and Sharma et al. (1981) elaborated history of jaundice in 7% of their patients. None of our patient had any major blood group in-compatibility. Raised levels of uncojugated Bilirubin in blood may cause its deposition in basal ganglia and other parts of brain, like Cerebellum and subcortical structures. These changes usually occur when serum level of unconjugated bilirubin is more than 18-20 mg%, however prematurity and anoxia accentuate bilirubin neurotoxicity (Diamond et al., 1966).

Post infective pathology is the next common factor in the causation of cerebral palsy. 16% (8) patients in our study material had post-infective etiology. Gauger (1951) observed 21.5%, Gibbs et al. (1963) 15%, Garg et al. (1965) 14.5%, and Sharma et al. (1981) reported 7% such cases. 6% (3) of them in our study had meningitis. Nearly similar figures had been reported by Garg et al. (1965) 8% and Sharma et al. (1981) 7%. Remaining 10% patients had encephalitis. Garg et al. also reported encephalitis in 6.4% cases. Various changes in cerebral vasculature, viz- vasculitis and arteritis cause infarection of cortical structure, supplied by affected vessel. The offending agent may also cause direct destruction (neuronal-necrosis) of

neurones but location of most severe damage varies, some cases having predominantly cortical involvement while in others basal ganglia bear the brunt.

In order to know the prevalance of various clinical types of cerebral palsy we classified our patients according to classification proposed by American Academy of cerebral palsy (Down and Hill 1980). Spastic variety had been the most common type of cerebral palsy so far observed by various workers. Its incidence is reported to vary from 40% (Phelps 1942) to 89% (Herlitz et al. 1955). 76% of our cases had spastic type of defect, thus our figures fairly coincide with others - Asher et al. (1950) 83%, Dundson (1952) 82%, Pertstein (1953), Woods (1957) 70%, Illingworth (1958) 72%, Mitchell (1959) 78.3%, Garg et al. (1960) 74.2%, Sharma et al. (1981, 82.7%). But it is noteworthy that Phelps (1942) reported very low incidence of spastic variety as he noticed athetosis to be equally common.

Next in the occurance was hypotonic variety which was diagnosed in 14% cases. Other workers reported it in 6.5% (Garg et al. 1965) and 5% (Sharma et al. 1981) patients. Relatively higher incidence of hypotonic type in present series could be the younger age of

type of cerebral palsy. Sharma et al (1981) also reported nearly similar number of such cases (7.3%). But Pohl (1950) and Illingworth (1958) reported lower incidence (2%) of these type of cases.

The reported incidence of convulsive disorders in cerebral palsy ranges from 68% (Yannet, 1944) to 15% (Pirrie et al., 1957). 56% cases in our study suffered from epilepsy. Patients having febrile convulsions were not included. Gauger (1951) noticed seizures in about 40% of his patients while respective values reported by other workers are - Perlstein et al. (1955) 47% and Garg et al. (1965) 23.4%. Our values are slightly higher and the possible explanation for which could be the younger age of our patients, as 44% of our patients were below 2 years of age. The younger patients are more commonly victimized by seizures (Perlstein et al., 1955).

If epilectics are further dichotomized, then we see that generalised tonic-clonic was the commonest seizure type, observed in 43% cases. Our this observation is further seconded by Pertstein et al. (1955) and Garg et al. (1965) who observed that type of seizure in 53% and 68% epileptics respectively. 10 (83%) out

our patients as some of them may later convert into spastic or athetoid type (Crothers and Paine 1959).

The indicence of athetosis reported so far, varies, from 40% (Phelps, 1942) to 2% (Herlitz and Redin 1955). We observed athetoid movements in only 1 (2%) case. But other workers reported higher incidence-Perlstein et al. (1955) 28%, Garg et al. (1965) 14.5%, Sharma et al. (1981) 4.1%. This low incidence in our study could be due to declining incidence of kernicterus and hyper bile rubinemia, as during earlier studies suitable management of neonatal jaundice like phototherapy and Exchange-transfusion, was not available. Phototherapy itself was first used by Cremer et al. in 1958.

2% (1) patient from present study material had ataxic type of cerebral palsy. Our figures are nearly similar to those reported by Sharma et al. (1981) who observed ataxia in 0.9% (2) of their patients, though Perlstein et al. (1935) observed 4% such cases. But it will not be wise to compare these cases due to small sample of our study.

3 patients (6%) had rigidity and tremors both in all the four limbs. They were categorized in mixed

of total 12 patients suffering from this variety of seizures, were spastics and still half of them (41.5%) had spastic diplegia. Fairly identical figures had been reported by Garg et al. (1965) where 75% patients of grand mal type had spasticity.

Next in the occurance was myoclonic seizure, as 18% of our patients exhibited this type only. Perlstein et al. (1955) observed myoclomus in only 7% of their material. The reason of this disparity could be the larger proportion of hypotonic patients in our study as they had highest incidence of such seizures (Bauer 1978).

We observed generalised tonic seizures in 10.7% patients. All the patients of generalised tonic type had spasticity. Perlstein et al. (1955) also had 14% patients of generalised epilepsy excepting grand mal type, in their study material and we included fairly similar number (14.2%) of patients of identical type in the form of generalised tonic (10.7%) and generalised clonic (3.7%) variety.

Incidence of Focal epilepsy was 10.7% in our patients. Two third patients of this variety had spasticity while remaining one third had hypotonia.

Perlstein et al. (1955) included 24% and Garg et al. (1965) included only 2.5% cases of focal epilepsy.

7.1% patients in this series had psychomotor epilepsy. They all presented with behavioural abnormality episodic onset. No one other than Peristein et al. (1955), observed psychomotor epilepsy who reported 0.4% such cases. This disparity can be due to associated behavioural problems itself in cerebral palsy which may easily be missed or discounted. Half of these patients had spastic diplegia and rest half had mixed type of cerebral palsy.

Rest 7.1% of our patients had mixed type seizures (more than one variety). We did not observed any patient of Petit mal type. 1.6% of Perlstein et al. (1955) series and 4.8% of Garg et al. (1965) series had Petit mal seizures.

It is important to compare these figures with general population. Kaushik et al. (1980) stated that out of all the types of epilepsy in general population, Grand-mal was the most common type, observed in 66.5% cases, followed by focal seizures (25.5%) Psychomotor (2%), petit mal (2%) and mixed epilepsy (4%). The lesser occurance of focal epilepsy in cerebral palsy could be

due to generalised, rather than localized, brain damage in these patients.

It can further be added that majority (75%) of epileptics belonged to spastic type of cerebral palsy or in other words 55% patients of the spastic group gave history of convulsions. Aird and Cohen (1950) included 65% and Perlstein et al. (1955) reported 60% epileptics. But Garg et al. (1965) reported that only 21.7% of all their spastic patients ever had seizures.

However incidence of seizures was highest in hypotonic patients i.e. 85.7%. Bauer (1978) also reported maximum incidence in atonic patients but his figures (69%) are lower than ours. It is important to mention here that all the patients of hypotonic diparesis had epilepsy in our series i.e. an incidence of 100%. None of our patients belonging to athetoid or ataxic category was epileptic.

Microcephaly is one of the commonest associated anomaly in the patients of cerebral palsy (Menkes, 1980; Brown and Fulford, 1984). 31 (62%) of our patients had microcephaly. The incidence of microcephaly was highest in hypotonic type as all of them (100%) had

microcephaly and next followed by spasticity where 55% had associated microcephaly.

Delayed milestones is the commonest presentation of cerebral palsy. Psychologists and pediatricians differ in their approach to developmental assessment. While psychologists tend to base their score on the ability to pass a certain test or make score, the pediatricians want to know, not just whether the child can achieve a particular skill but the maturity of his response and therefore how long he has been able to do it. The clinicns basic aim is to determine how far the child has developed in relation to normal. One of the methods of expression of development, for the purpose of comparison, is to assess child's Developmental Quotient (D.Q.). Thus we have calculated developmental quotient of each of fifty patients according to methos described by Prabhakar and Kumar (1983).

Average developmental quotient of our study was 34.9%. Maximum developmental retardation was observed in Athetosis (D.Q. = 11%) and spastic triparesis (D.Q. 19%). Though much emphasis can't

be imparted to these figures as one patient only belonged to these categories.

Next in the sequence of developmental retardation are Hypotonic, (D.Q. = 25.14%) and spastic Quadriparetic (D.Q. = 26.6%) patients. While least developmental retardation was seen in ataxic (D.Q. = 65%), Spastic (left) hemiparetic (D.Q. = 59.7%) and Spastic (left crural) monoparetic (D.Q. = 57.0%) patients. Spastic diparetics which comprised the substantial amount of present study material, had developmental quotient of 33.3%, and overall D.Q. in all the spastics was 36.9%.

Electroencephalographic abnormalities are quiet frequent in cerebral palsied children, but these abnormalities become more apparent and frequent if epilepsy is also associated. All the patients were sedated before hand as they were not cooperative. However no appreciable difference is noted in sleep and awake recordings (Gauger, 1951).

Reported values of abnormal EEG tracings in cerebral palsy varies from 65% (Aird et al. 1950, Perlstein et al. 1955) to 80% (Gauger 1951). However

60% of our patients had essentially abnormal EEG records. Bauer (1978) found fairly similar number of patients (61.3%) having abnormal electroencephalograms.

If these patients with abnormal electroencephalograms are further subclassified then it had been observed that maximum abnormality is seen in spastic patients. Aird and Cohen (1950) noted 88% EEG abnormality in his study material and Perlstein et al. (1955) noted it in about 72% patients. Though still lower figures (61%) had been observed by Bauer (1978). 55% of our spastics had abnormal electroencephalograms.

In contrast, we recorded maximum EEG abnormality in Hypotonic patients (70%) but our figures fairly coincide with that of Bauer (1978) corresponding figure is 71%. Moreover 66.6% of mixed type of cerebral palsy in our study had abnormal EEG. But it is important to note here that mixed type of cerebral palsy was observed in only 6% (3) patients by us. So it will be difficult to comment on them. Same holds true for ataxic and athetoid patients. None of the athetoid patients (2%) had any electroencephalographic abnormality.

This seems reasonable, because athetosis is caused by extrapyramidal lesions, and injuries to extrapyramidal structures (being deep seated) do not usually produce abnormality in electroencephalograms.

Epilepsy, being an episodic electrical abnormality of brain, quiet frequently gives rise to some electroencephalographic abnormality, either in the form of inter-ictal discharges or as a menifestation of ictal- brain damage. Out of all the 60% patients having abnormal electroencephalograms, only two third had epilepsy while rest had EEG abnormality without ever experiencing seizures. Gibbs et al., (1963) however observed that normal electroencephalograms are far more common among non-epileptics than epileptics. In our study incidence of abnormal EEG in epileptics was 71% while among non epileptics only 30% had abnormal electroencephalograms. But the general charactor of electroencephalographic findings in epileptic and non epileptic groups in much the same, except for much higher incidence of normal records in non-seizure group while much frequent occurance of spike foci in seizure group.

High degree of concordance exists between laterality of clinical involvement and the laterality of electroencephalographic abnormality in cerebral palsy. In symmetrical forms of cerebral palsy, the electroencephalographic abnormalities when present are usually bilaterally symmetrical but in asymmetric forms, EEG findings are either unilateral or predominantly on contralateral side (Perlstein et al. 1955). About one quarter (26.6%) of our patients had focal changes in EEG and in 62.5% of these cases, the lateralization was correct i.e. lateralized to the side opposite of motor deficit. Maximum lateralization was seen in hemiparetic patients as all (100%) the patients with left hemiparesis revealed right hemispherical damage while two third patients of right hemiparesis had only left sided and rest one third had predominantly left sided abnormalities. None of our patients with bilateral affection clinically, had asymmetrical EEG. Reduced voltage production and asymmetry of sleep spindles were the most significant abnormalities in lateralising EEG, Gibbs et al. (1963)noted lateralization in 43% cases and out of that, in 97% the lateralization was correct. But this should not mean that brain damage is exclusively unilateral in hemiplegic patients (Gibbs et al. 1963).

In most of the patients there was evidence of intermittent generalised slowing suggestive of diffuse brain damage while next common abnormality was spikes or spiks-and-slow-wave complexes which however were common in epileptics.

In the last we have tried to compare EEG abnormality and developmental quotient. In our series average developmental quotient was 34.9% and majority of our patients (76%) had developmental quotient below 50%. The developmental retardation was more severe in the patients with abnormal EEG than in the patients with normal EEG. This was statistically significant as 'p' value is <0.05. This suggests that more is the developmental-retardation, more are the chances of abnormality in electroencephalograms. We could not find any study correlating developmental quotient and EEG findings cerebral palsy but studies conducted, where, intelligence-quotient has been correlated with EEG finding in these patients, report that more severe the mental retardation more are the chances of EEG abnormality (Gauger 1957, Gibbs et al. 1963). We also observed significantly more

chances of developmental retardation in epilepties, as of all the epilepties more than 85% had D.Q. below 50%. This could be due to further brain damage in epilepties (Lennox 1942, Waterlain 1978).

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SUMMARY AND CONCLUSIONS

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The present project work entitled, "Electro-encephalography and epilepsy in cerebral palsy" was performed over 50 patients of cerebral palsy. Every patient was classified according to the classification proposed by American Academy of cerebral palsy (Down and Hill, 1980). Detailed perinatal history and history of events during infancy and childhood, upto the age of 5 years, was then asked for, from the mother of the patient. Special emphasis was imparted to the history of seizures. Details of developmental mile stones were asked in every case individually. Complete physical examination was performed in every case with special emphasis to neurological examination. Fundus examination and X-ray skull, both A.P. and lateral views were done in all the cases. Routine electroencephalographic recording was done in every patient, who was sedated before hand. Observations were tabulated and data analysed.

Age of the patients ranged from 7 months to 10 years. Males dominated in this study and male: female ratio was 16:9. Perinatal factors were most important in the causation of cerebral palsy notably

Birth Asphyxia (44%) and Prematurity (30%). Among the events of infancy and childhood. Post-infective pathology (16%) was the most common precipitating factor.

The sample was dominated by spastic patients who constituated the bulk (76%) of this series. Among them diparesis was the commonest motor defect as they only comprised of nearly half (46%) of the total study material. Hypotonic cerebral palsy was the next commonest type, observed by us in 14% cases. Athetosis and ataxic forms were found to be rare.

Epilepsy is a fairly common problem as 56% patients, ever experienced seizures in their life. The clinical type of seizure, observed by us, in descending order was - generalised tonic-clonic (43%), Myoclonus (18%), generalised tonic (10.7%), focal (10.7%), Psychomotor (7.1%), mixed (7.1%) and generalised clonic (3.5%). Incidence of seizures was highest in hypotonic type in which 85.7% had epilepsy. This was followed by spastic variety, where 55% were epileptics.

Developmental retardation is a very important menifestation of cerebral palsy, as mean developmental quotient (D.Q.) of our patients was 34.9%, with maximum retardation in hypotonic cerebral palsy. More or less, similar extent of developmental retardation had been observed in spastic triparesis and athetosis but much emphasis could not be given to them as both these types were represented by 1 (2%) patient only. Least retardation of development was seen in spastic hemiparesis (Left).

60% of our patients had abnormal electro-encephalograms. Spastic patients had quiet fair (55%) chances of EEG abnormality though maximum chances of EEG abnormality were in hyptonic patients as 70% of these patients had abnormal EEGs. The EEG defects are uncommon/rare in ataxia and athetosis. Out of all the patients having abnormal EEG, one third no seizures at all. Quiet a common (26.6%) number of patients had focal changes in EEG and out of them, in 62-5% cases the lateralization was correct i.e. changes were seen in the opposite hemisphere. Out of all the patients having EEG defects, 66.6% had Developmental Quotient below the mean value (34.9%). We also observed that chances of EEG-abnormality were more, in more severe developmental retardation. Also, epileptecs had quiet

a significant developmental retardation as 85% of them had D.Q. below 50%.

Following conclusions could be drawn from present study:-

1- Males are predominantly affected as male: female ratio was 16:9.

2- Birth Asphyxia and Prematurity are the two most common etiological factors as these only were responsible for two third cases.

3- Spastic out number any other clinical type of cerebral palsy as 76% patients belonged to this group.

4- Seizure disorders are very frequent in cerebral palsied children as more than half (56%) had associated epilepsy. Hypotonic type in general and hypotonic diparesis in particular are more commonly complicated by epilepsy. The commonest type of seizure disorder is generalised tonic-clonic (43%) followed by Myoclonus (18%).

5- Developmental Retardation is usually always there in cerebral palsy as mean development quotient was 34.9%. Maximum developmental

retardation was apparent in hypotonic cerebral palsy while least retardation was a feature of spastic hemiparesis (Left).

6- Electroencephalographic abnormalities are quiet frequent in cerebral palsy as 60% patients were having essentially abnormal records. Chances of EEG abnormality are more if epilepsy is associated. Moreover chances of EEG-abnormality are more in hypotonic cerebral palsy followed by spastic variety.

7- High degree of concordance exists between laterality of clinical findings and EEG findings. Asymmetry constitutes the most significant EEG-abnormality in asymmetrical types of cerebral palsy like hemiparesis. In these electroencephalograms, amplitude seems to have more localising value than frequency. Suppression of voltage production, especially reduced voltage of spindles is reliable lateralising sign.

8- One should very cautiously look for epilepsy in these patients as pathophysiological state which underlies epilepsy does not necessarily

express itself in major convulsions or in obvious clinical seizures. The minor menifestations of epilepsy, if present are likely to be overlooked or discounted.

9- Higher is the developmental retardation, more are the chances of EEG-abnormality.

10- Electroencephalography adds another dimension to our view of cerebral palsy. In combination with other parameters of evaluation, it is useful for diagnosis and prognosis.

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